Fundamentals of Maxillofacial Surgery

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Editor

Fundamentals of Maxillofacial Surgery

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Preface

The development of a knowledge base is a gradual process; mastery of a subject demands much time and energy. In the course of their education, young surgeons are exposed to many facts in their reading and in discussions with peers. Their assimilation and integration of this factual material is later tested by objective means in the operating room.

This book is designed to give a comprehensive review of maxillofacial surgery from many perspectives, such as anatomy and embryology, it is not intended to be a research base. The facts are brought together in a progressively developed text that is designed to help the reader understand many, but certainly not all, of the principles of maxillofacial surgery.

Entitled the *Fundamentals of Maxillofacial Surgery*, it brings together information on a multitude of subjects, all of which deal with different facets of maxillofacial surgery. Although this material appears throughout the literature, this is the only book in which these topics are brought together. The book begins with the embryology and anatomy of the head, neck, and craniofacial regions. It is necessary to understand the normal anatomy of these regions in order to correct the anatomy that presents abnormal shapes, positions, and incomplete development. The next two chapters, odontogenesis and oral anatomy, cover topics that are absent or inadequately taught in the medical school curriculum. To deal with the jaws and their associated teeth, a knowledge of the basics of dental anatomy is imperative. Unfortunately, students cannot apply the basics of anatomy until they are in a clinical setting. The use of some of the dental anatomy discussed is therefore applied in the chapter on "Local Anesthesia and Infiltration Techniques" and in other chapters dealing with the teeth and their position in the masticatory apparatus.

Chapters 8 through 12 examine the evaluation and treatment of maxillofacial trauma. This portion of text deals with the basic tenets in the treatment of facial fractures in adults and children. The techniques of wiring made popular by Milton Adams, MD in the 1940s and the use of Ivy loops, Gilmer wire, and the continuous stout wire ligature techniques are presented. We are aware that many of these techniques are now of historical importance only. But, knowledge of past techniques may sometimes help us to deal with a problem that more up-to-data techniques of plates and screws cannot solve. A general discussion of the facial trauma patient is also presented with a thorough, but by no means complete, discussion of the treatment of maxillary mandibular and mid-facial fractures. It is hoped that with this information, readers will be able to treat most of the cases of maxillofacial trauma to which they will be exposed.

Chapters 13 through 21 discuss the evaluation of treatment of the patient with certain orthognathic and temporomandibular joint surgical problems. These chapters are designed to present methods of case assessment and treatment planning, but do not represent the only—or even, in some cases, the most widely used—treatment plans or cephalometric evaluation techniques. These chapters provide a guide to tried-and-true operative techniques that should help the reader appropriately deal with the patient's problems and needs. These chapters do not offer an exhaustive treatise on all known techniques. Rather, they offer the knowledge of the particular authors and their individual experiences in the field of maxillofacial-orthognathic surgery. As such, these chapters reflect each author's built-in biases and prejudices.

The final two chapters deal with the materials necessary to set up an office dental laboratory for the surgeon specializing in this area. It also discusses the techniques necessary to take impressions, pour models, and place finished models on the articulator. This is followed by a discussion in the construction of acrylic splints, with the use of clinical examples where necessary. Chapter 23 shows how splints can be an aid in the treatment of fractures and orthognathic surgical cases when used with the more modern techniques of rigid fixation.

Finally, I would like to thank all of my contributing authors for their efforts in this enormous undertaking. In particular, I would like to thank Dr. Irving Polayes, for without his pioneering efforts this book could never have been written. It is my hope that the *Fundamentals of Maxillofacial Surgery* will help my medical colleagues deal effectively with this area of surgical correction.

James W. Ferraro

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CHAPTER 1

Embryology of the Head and Neck

Steven R. Cohen

Prenatal Development

Human development begins at fertilization. Fusion of the two haploid germ cells produces a *zygote*, a diploid cell with 46 chromosomes, the usual number for a human being. Initiation of cleavage follows fertilization and results in a series of rapid mitotic cell divisions within the zygote. About 30 hours after fertilization, division of the zygote into two daughter cells (called blastomeres) occurs.[1,2] The morula is a solid ball of 12 to 16 blastomeres and is formed about 3 days after fertilization.[1,2] Four days after fertilization, the compact mass of cells forming the morula begins to develop fluid-filled cavities.[1,2] The fluid increases, separating the cells into two layers. The outer layer, which gives rise to the placenta, is called the trophoblast (from the Greek word *trophe*, meaning nutrition). The centrally located cells, known as the inner cell mass, give rise to the embryo[1,2] (Fig. 1.1).

During the second week, morphological changes occur in the inner cell mass, producing a bilaminar embryonic disc (Figs. 1.2 and 1.3). It is at this time that the prochordal plate develops as a localized thickening of the hypoblast, presaging the future cranial region and mouth of the embryo[1] (Fig. 1.4).

The third week is characterized by formation of the primitive streak. Gastrulation begins on approximately Day 14 and ends at approximately Day 19.[1,2] It is through this process that the bilaminar embryonic disc is converted into a trilaminar structure. Formation of the primitive streak and the notochord area are among the important events that occur during gastrulation. As the primitive streak elongates, its cranial end thickens to form a primitive knot (Fig. 1.5). It is at the time of primitive streak appearance that it becomes possible to identify the craniocaudal orientation of the embryo.[1] At about 16 days, the intraembryonic mesoderm begins to appear.[1] Mesoblastic cells then migrate cranially from the primitive knot to form a midline cord known as the notochordal process (Fig. 1.6), which grows until it reaches the prochordal plate (Fig. 1.7). The notochord moves toward the prochordal plate, stopping at the overlying ectoderm, which forms the oropharyngeal membrane.

As the notochord develops, the embryonic ectoderm over it thickens, forming the neural plate. The ectoderm of the neural plate, called neuroectoderm, gives rise to the central nervous system (consisting of the brain and spinal cord). The neural plate first appears cranial to the primitive knot and dorsal to the notochordal process.[1] As the notochord elongates, the neural plate broadens and eventually extends cranially as far as the oropharyngeal membrane. On approximately Day 18, the neural plate evaginates, forming a neural groove with neural folds on each side.[1] By the end of the third week the neural folds begin to fuse, converting the plate into a tubular structure.[1] Once it separates from the surface ectoderm, the neural tube fuses. As fusion occurs, some ectodermal cells lying along the crest of each neural fold lose their epithelial affinities and attachments and migrate inward. They appear as an irregular flattened mass called the neural crest. Neural crest cells give rise to the spinal ganglia and the ganglia of the autonomic nervous system. The ganglia of the cranial nerves (V, VII, IX, and X) are also partly derived from the neural crest.[1]

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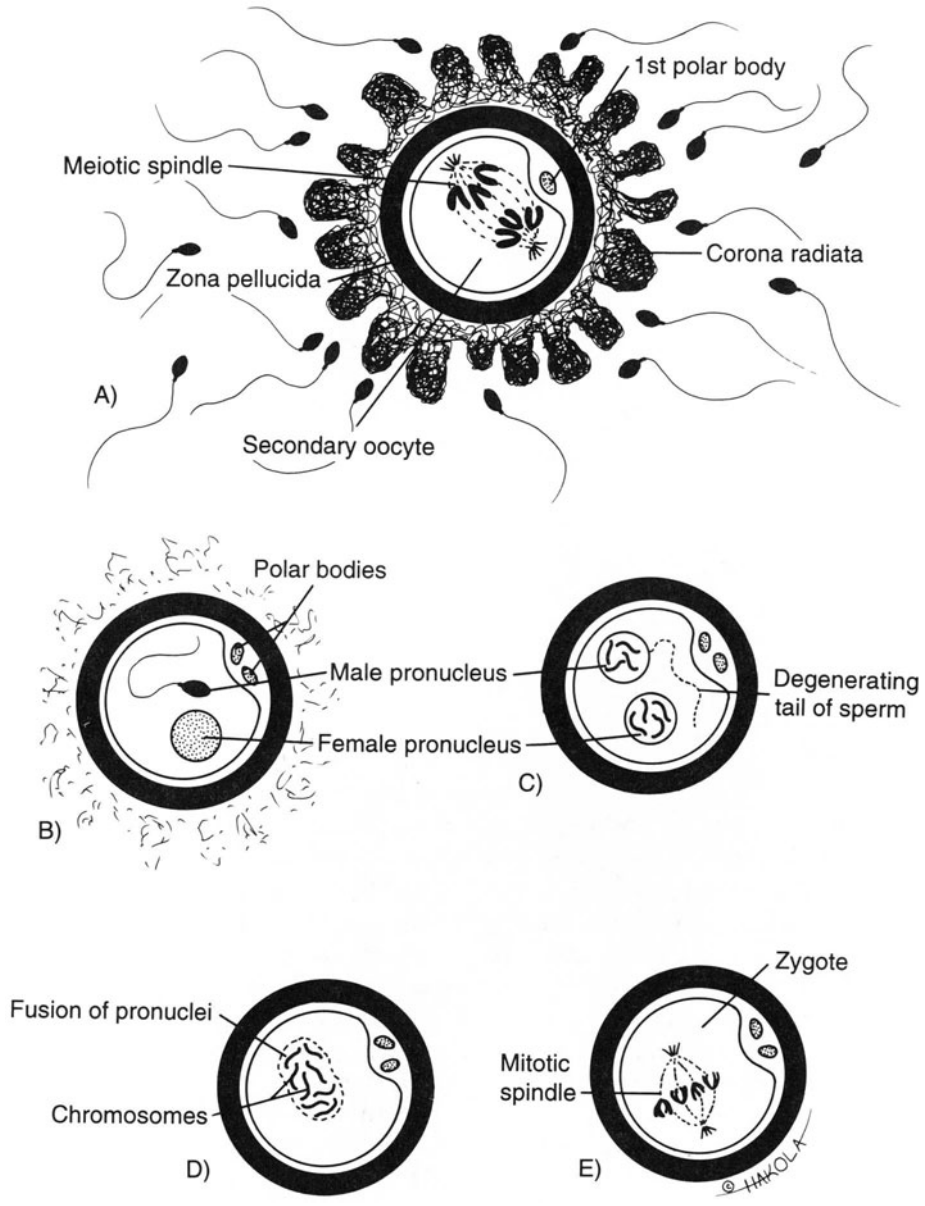

Figure 1.1. Artist's drawings depicting fertilization, which begins when the sperm contacts the secondary oocyte's plasma membrane and ends with the intermingling of maternal and paternal chromosomes at the metaphase of the 1st mitotic division of the zygote. (A) Secondary oocyte surrounded by sperm. (B) Corona radiata has disappeared; a sperm has entered the oocyte, and the second meiotic division has occurred forming a mature ovum. (C) The sperm head has enlarged to form the male pronucleus. (D) The pronuclei fuse. (E) The chromosomes of the zygote are arranged on the mitotic spindle in preparation for the first cleavage.

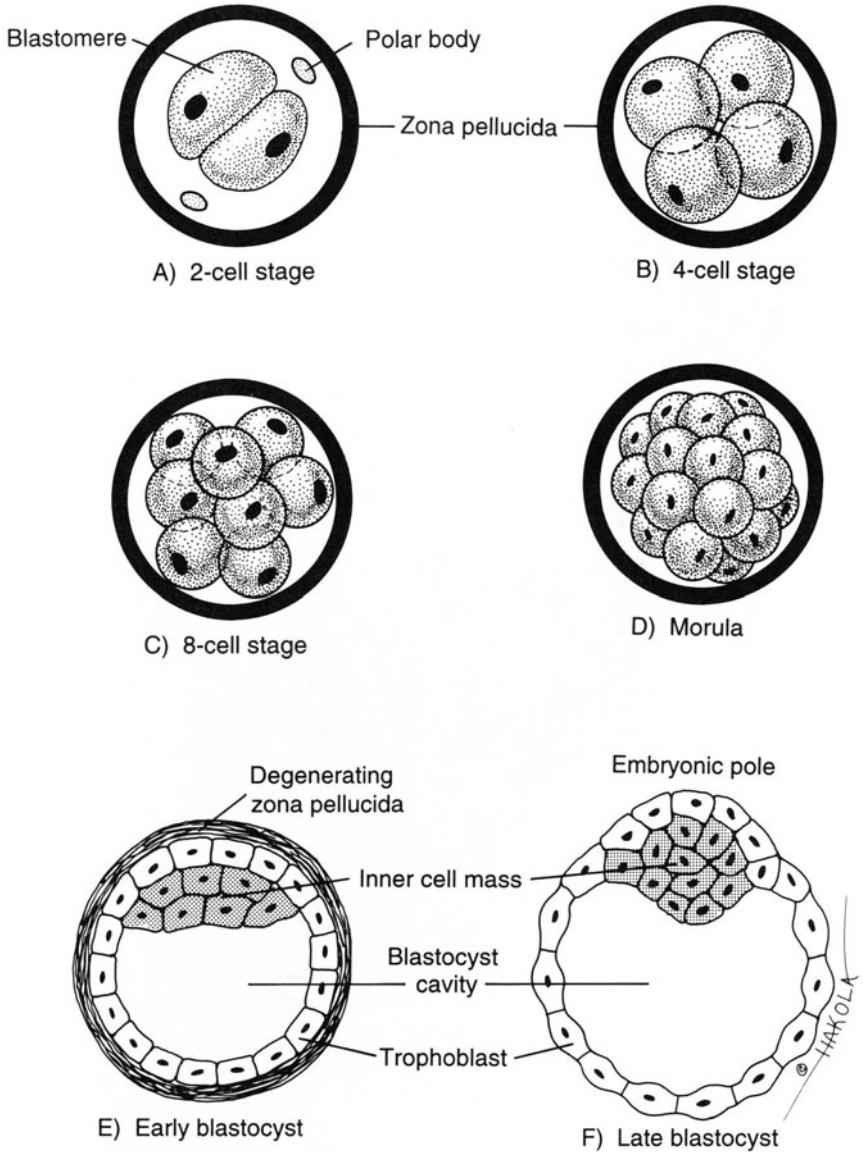

Figure 1.2. Artist's depiction of zygote cleavage and formation of the blastocyst. (A–D) Various stages of cleavage until morula forms at the 12–16 cell stage. (E, F) Sections of blastocysts. Note the blastocyst cavity and the inner cell mass.

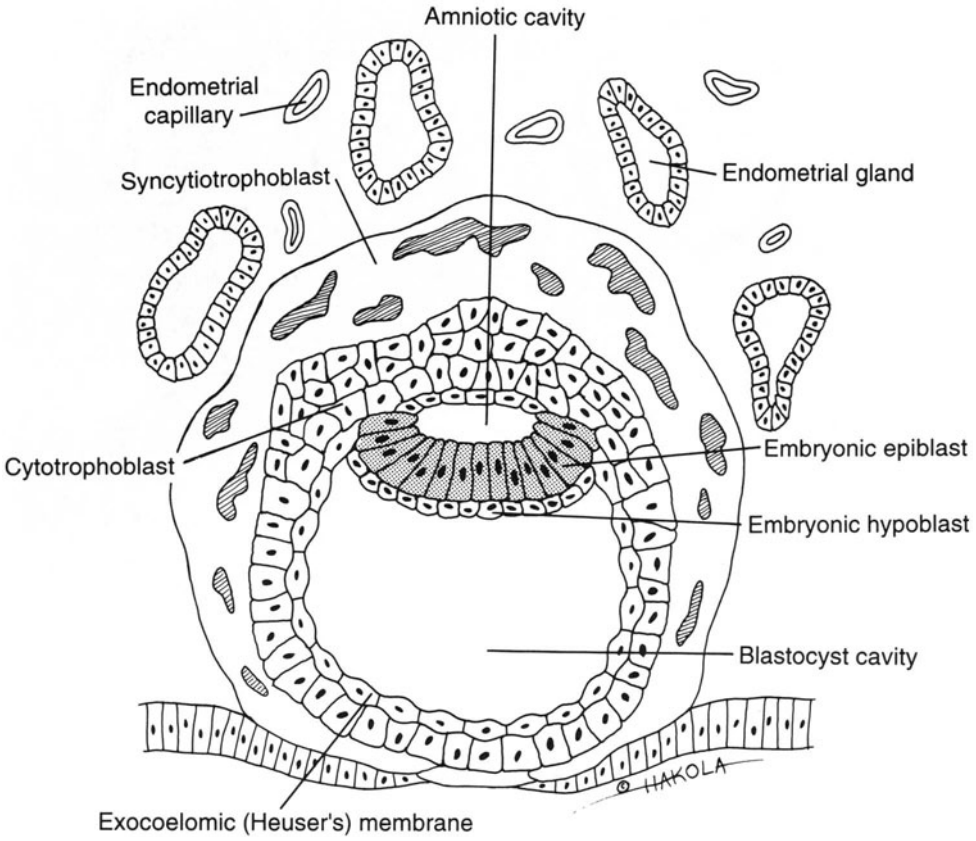

Figure 1.3. Artist's depiction of the bilaminar embryonic disc, which is composed of epiblast and hypoblast. The epiblast gives rise to all three germ layers of the embryo (ectoderm, mesoderm, and endoderm). The hypoblast represents the primitive embryonic endoderm, which gives rise to the extra-embryonic region such as the embryonic cavity and yolk sac.

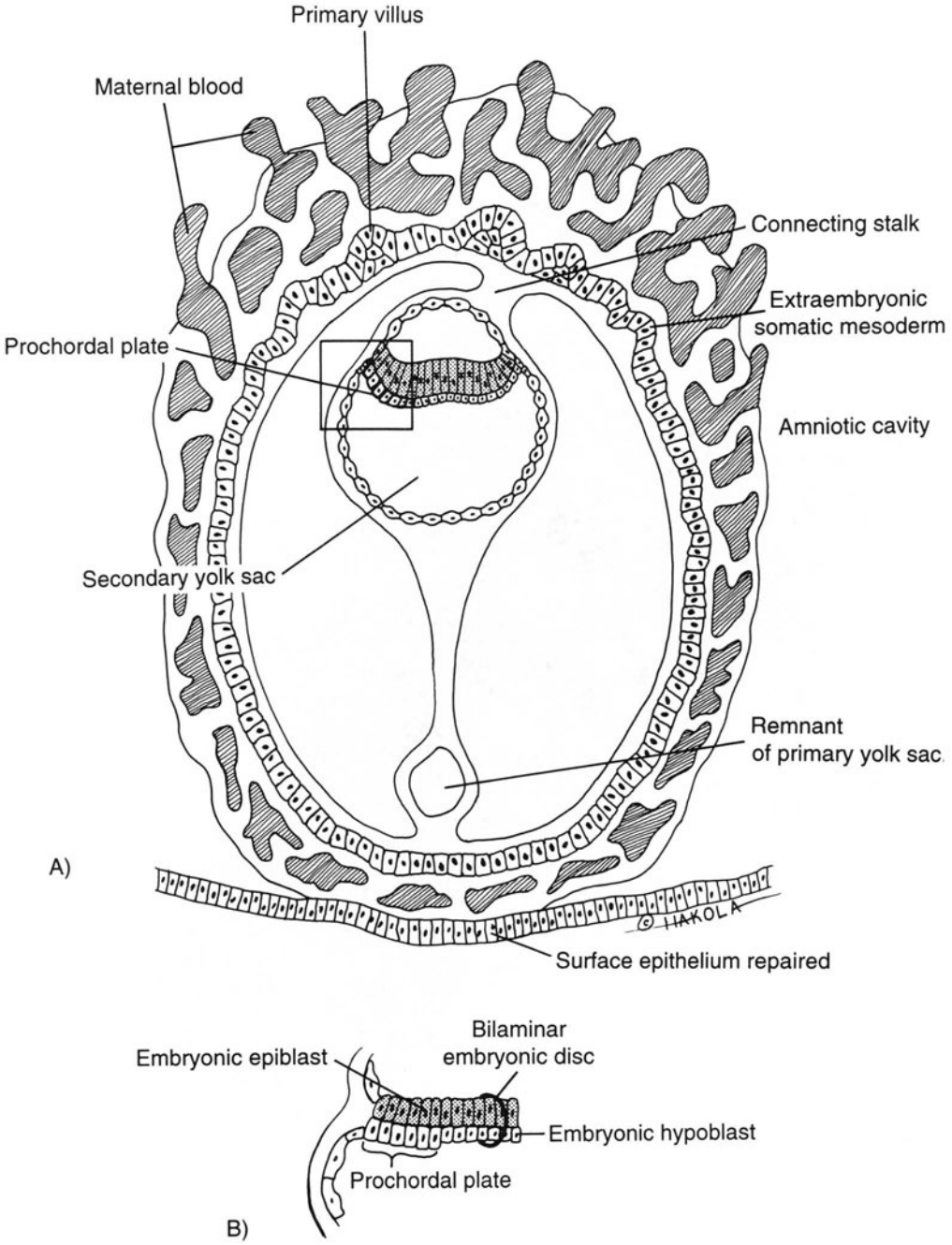

Figure 1.4. The prochordal plate develops as a localized thickening of the hypoblast, presaging the future cranial region and mouth of the embryo.

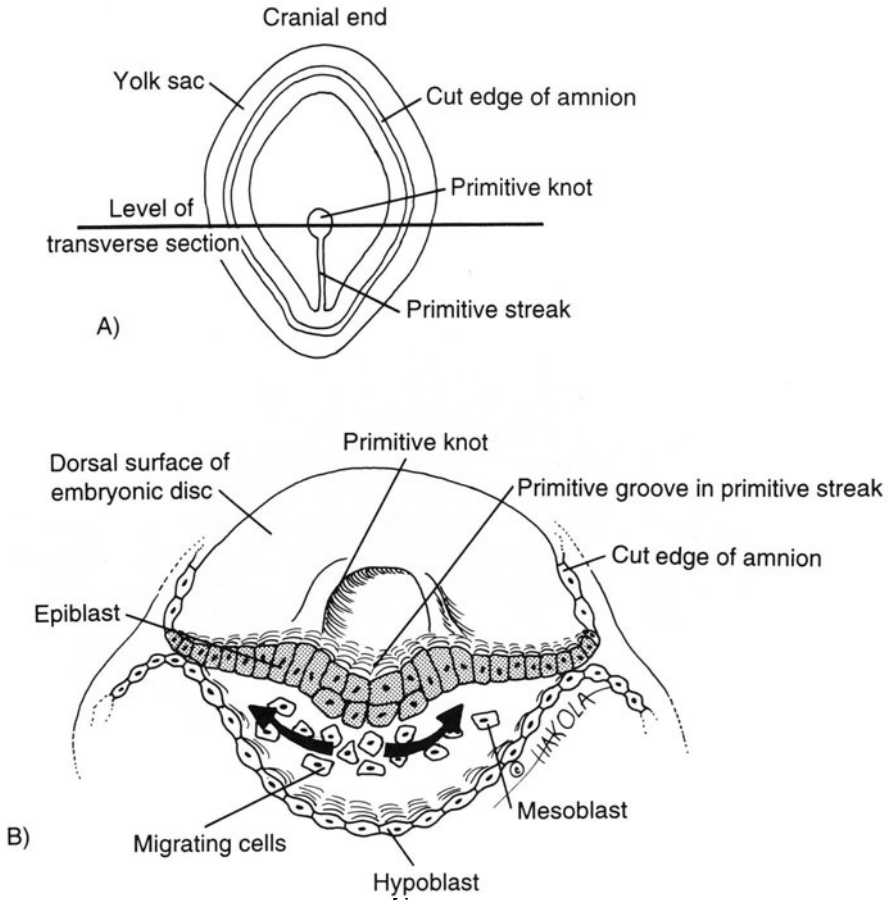

Figure 1.5. Formation of the primitive streak and notochord are important events that occur during gestation. As the primitive streak elongates, its cranial end thickens to form a primitive knot. Shown here is the drawing of the cranial half of the embryonic disc during the third week. The disc has been cut transversly to show the migration of mesenchymal cells from the primitive streak.

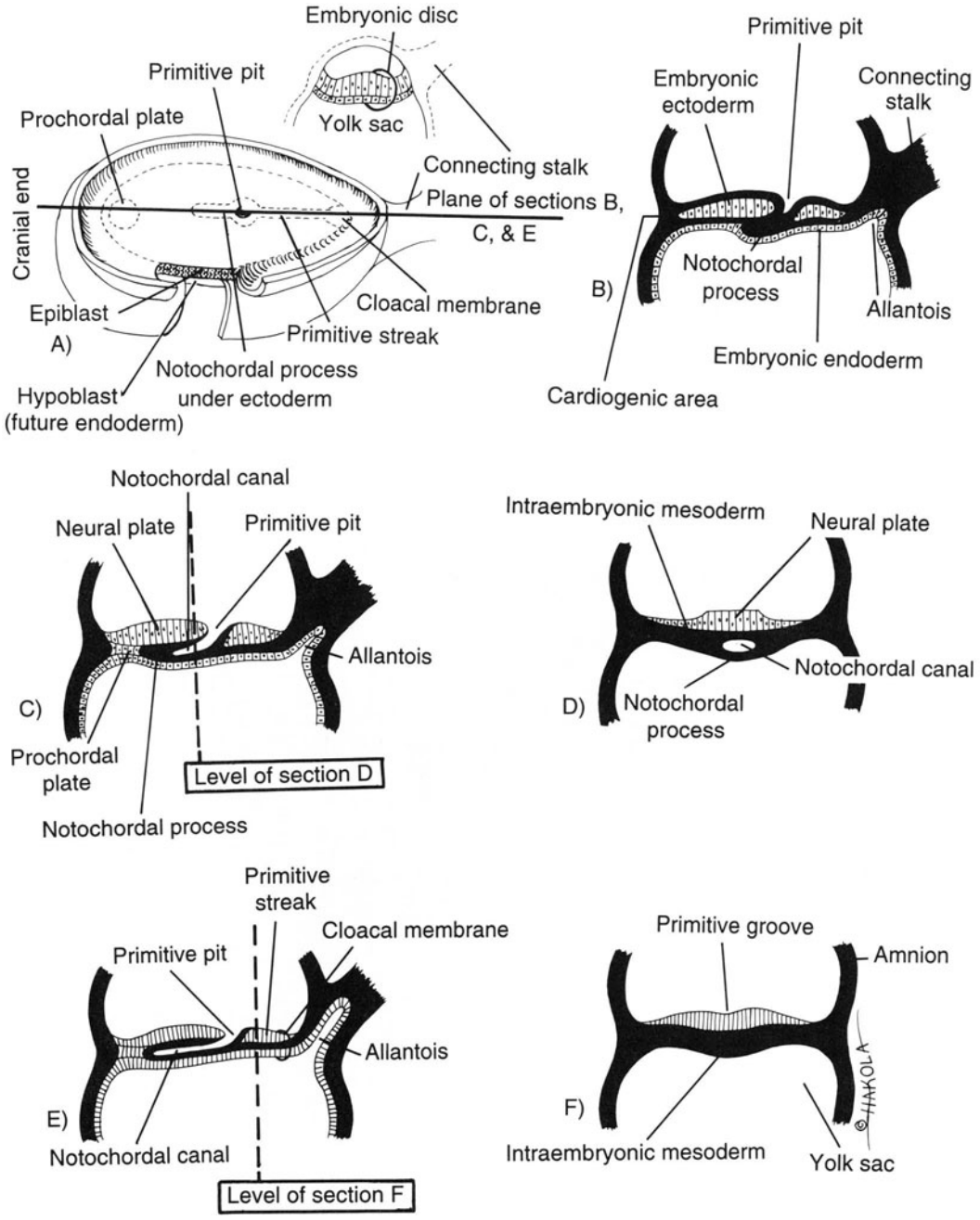

Figure 1.6. (A) Sagittal section showing notochordal canal growing toward the prochordal plate. (B) Transverse section through embryonic disc at the same level.

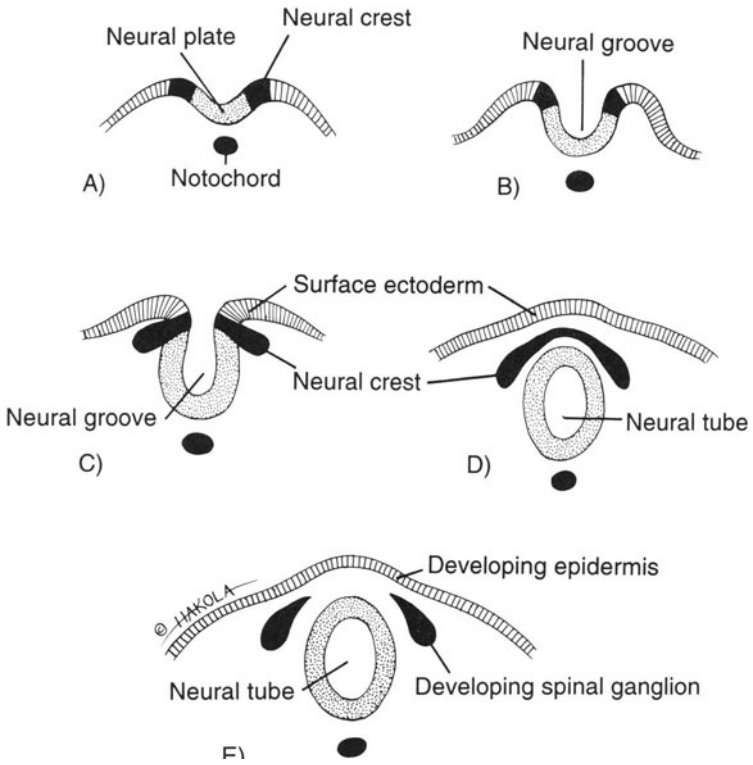

Figure 1.7. Illustration of transverse sections through progressively older embryo. The formation of the neural groove (B, C), neural tube (D, E), and neural crest (A) up to the end of the fourth week are shown.

The Embryonic Period (Fourth to Eighth Weeks)

By the end of the embryonic period, the major organ systems have begun to develop. Gradually, the forebrain grows cranially beyond the oropharyngeal membrane to overhang the permanent heart (Fig. 1.8). The primitive germ layers (embryonic ectoderm, mesoderm, and endoderm) give rise to all the tissues and organs of the embryo (Fig. 1.9). The ectoderm gives rise to the central nervous system; the peripheral nervous system; the sensory epithelia of the eye, the ear, and the nose; the epidermis and its appendages; the mammary glands; the pituitary gland; the subcutaneous glands; and the enamel of the teeth. The neural crest cells, which are derived from ectoderm, give rise to cells of the spinal, cranial, and autonomic ganglia; sheathing cells of the peripheral nervous system; pigment cells of the dermis, muscle, connective tissue, and bone of the branchial arch origin; the suprarenal medulla; and the leptomeninges.

The mesoderm gives rise to cartilage, bone, and connective tissue; striated and smooth muscles; the heart, blood, and lymph vessels and cells; the kidneys; the gonads and the genital ducts; the serous membranes lining the body cavity; the cortex of the body cavity and the cortex of the suprarenal gland. The endoderm layer gives rise to the epithelial lining of the gastrointestinal and respiratory tracts, the thyroid gland, the parathyroid glands, the sinuses, the liver, the pancreas, the epithelial lining of the urinary bladder and urethra, the epithelial lining of the tympanic cavity, the tympanic antrum, and the auditory tube.

Development depends on a precisely coordinated interaction among many genetic environmental factors. Tissue interactions, regulated migrations of cells and cell colonies, controlled proliferations, and cell death are some of the control mechanisms that guide differentiation and ensure synchronized development.[1]

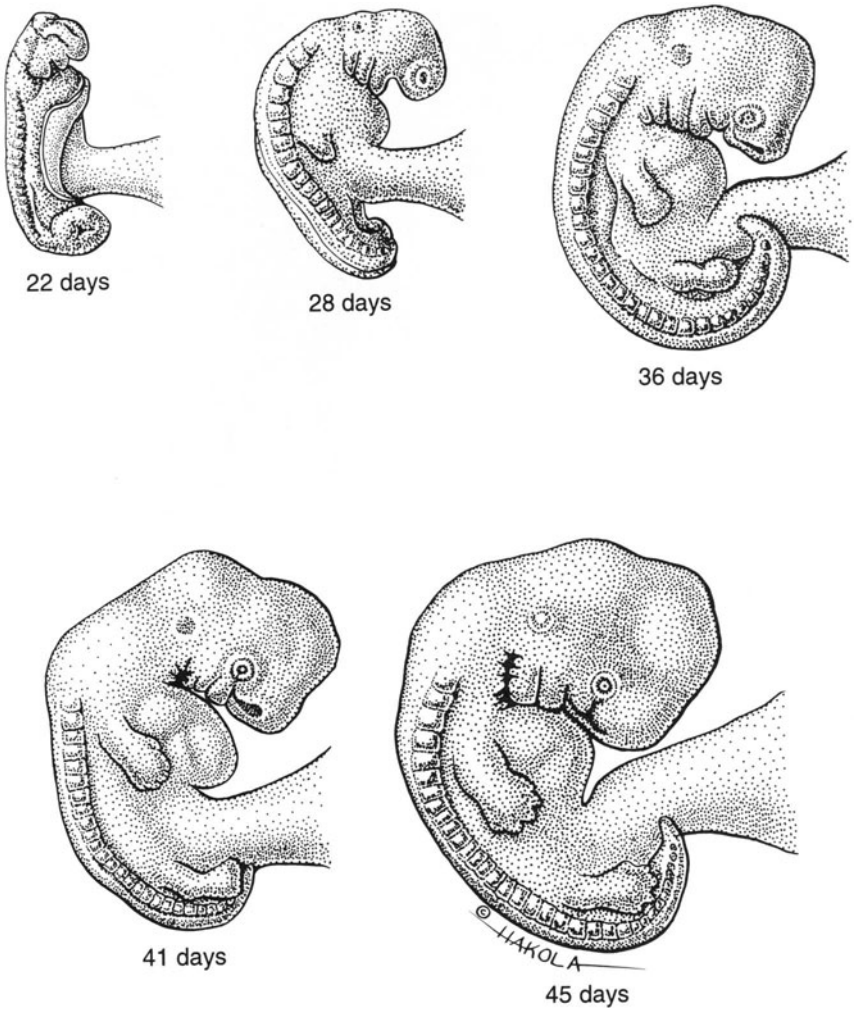

FIGURE 1.8. Gradual enlargement of forebrain and formation of branchial arches.

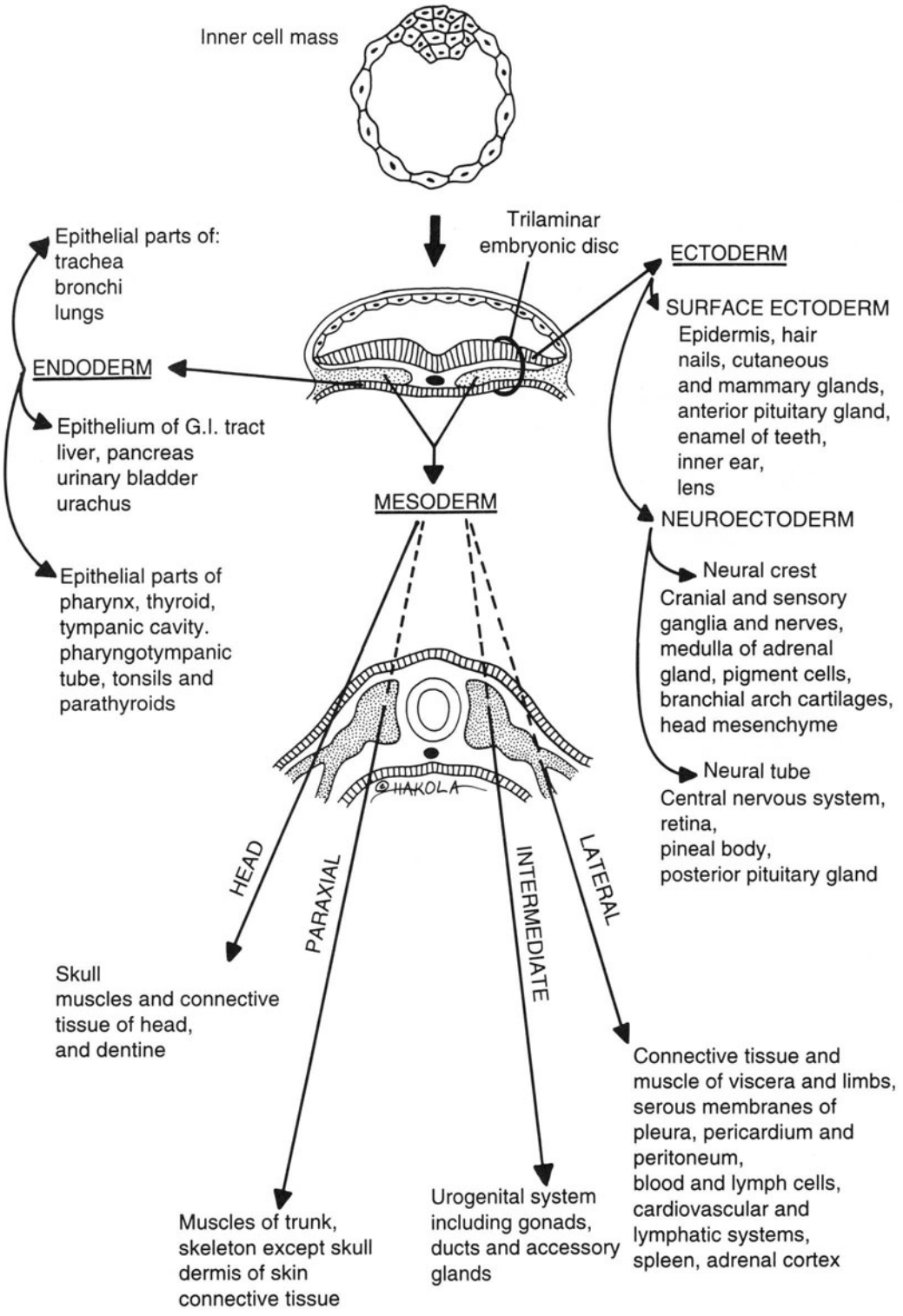

Figure 1.9. Origin and derivatives of the three germ layers.

The Fetal Period (Ninth Week to Birth)

The transition from embryo to fetus is gradual. Development during the fetal period is primarily concerned with growth and differentiation of the tissues and organs (Fig. 1.10).

Development of the Face

The five facial primordia appear around the stomadeum, or permanent mouth, early in the fourth week. The frontonasal prominence is formed by proliferation of mesenchyme ventral to the forebrain and constitutes the cranial boundary of the stomadeum. Paired maxillary prominences from the first branchial arch form the lateral boundaries, while the paired mandibular prominences from the same arch constitute the caudal boundary of the stomodeum (see Fig. 1.11).

The development of the face occurs mainly between the fifth and eighth weeks. The mandible is the first part of the face to form. This results from merging of the medial ends of the two mandibular prominences during the fourth week. By the end of the fourth week, bilateral oval-shaped thickenings of the surface ectoderm, called nasal placodes, develop on each side of the lower aspect of the frontonasal prominence.[1] Mesenchyme proliferating at the margin of these placodes produces horseshoe-shaped elevations called the medial and lateral nasal prominences. The nasal placodes now lie in depressions called nasal pits (Fig. 1.11A).

By the end of the fifth week, the eyes are slightly forward on the face and the external ears have begun to develop. By this time, each maxillary prominence has merged with the lateral nasal prominence along the line of the nasal lacrimal groove. This establishes continuity between the side of the nose, formed by the lateral nasal prominence, and the upper cheek region formed by the maxillary prominence (Fig. 1.11B). At the same time, between the 6th and 12th weeks, the palate develops and fuses from anterior to posterior (Fig. 1.11C).

Figure 1.10. Artist's rendition of fetal growth in weeks.

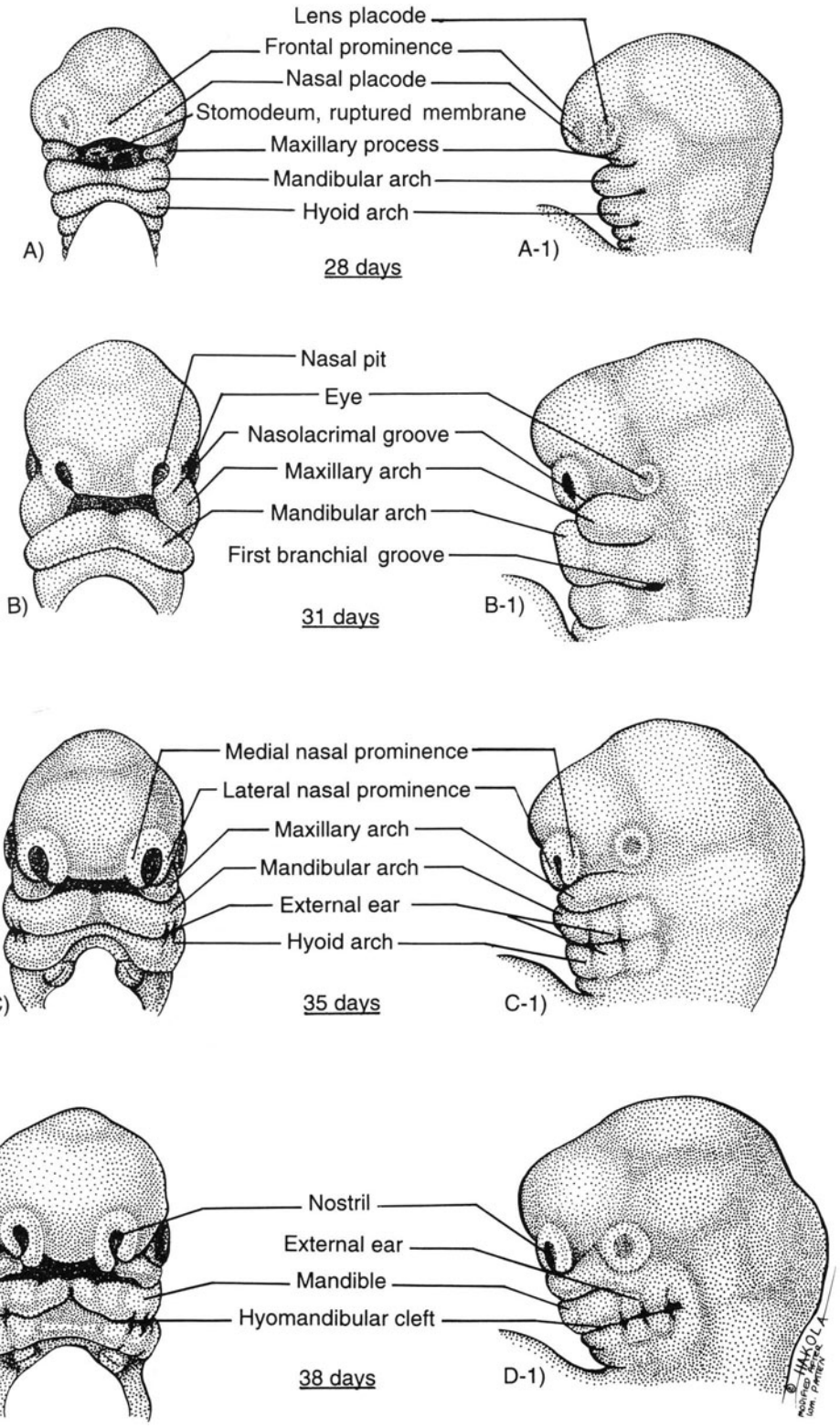

Figure 1.11. (A) Progressive stages of development in the human face.

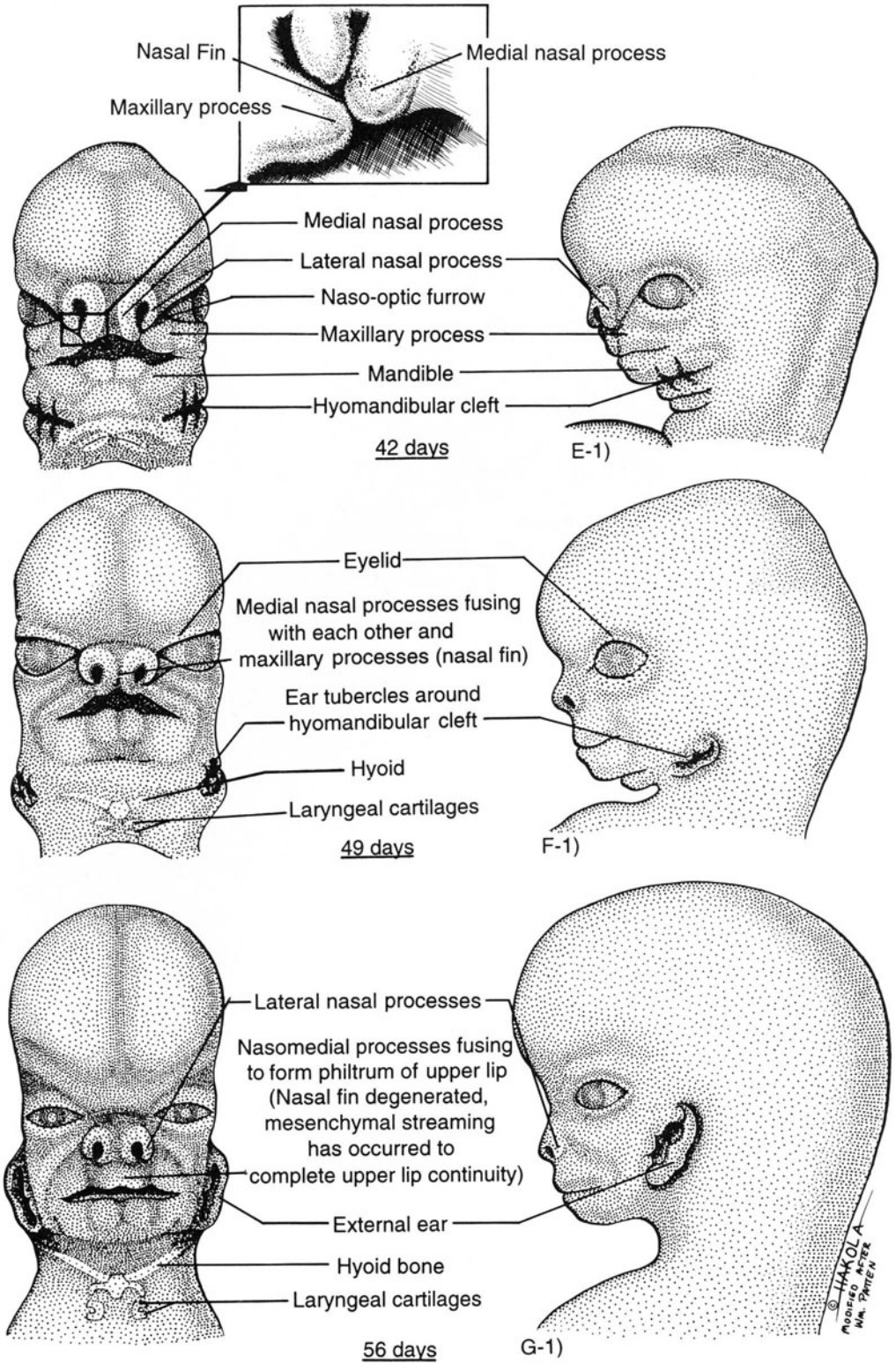

Figure 1.11. (B) Progressive stages of development in the human face.

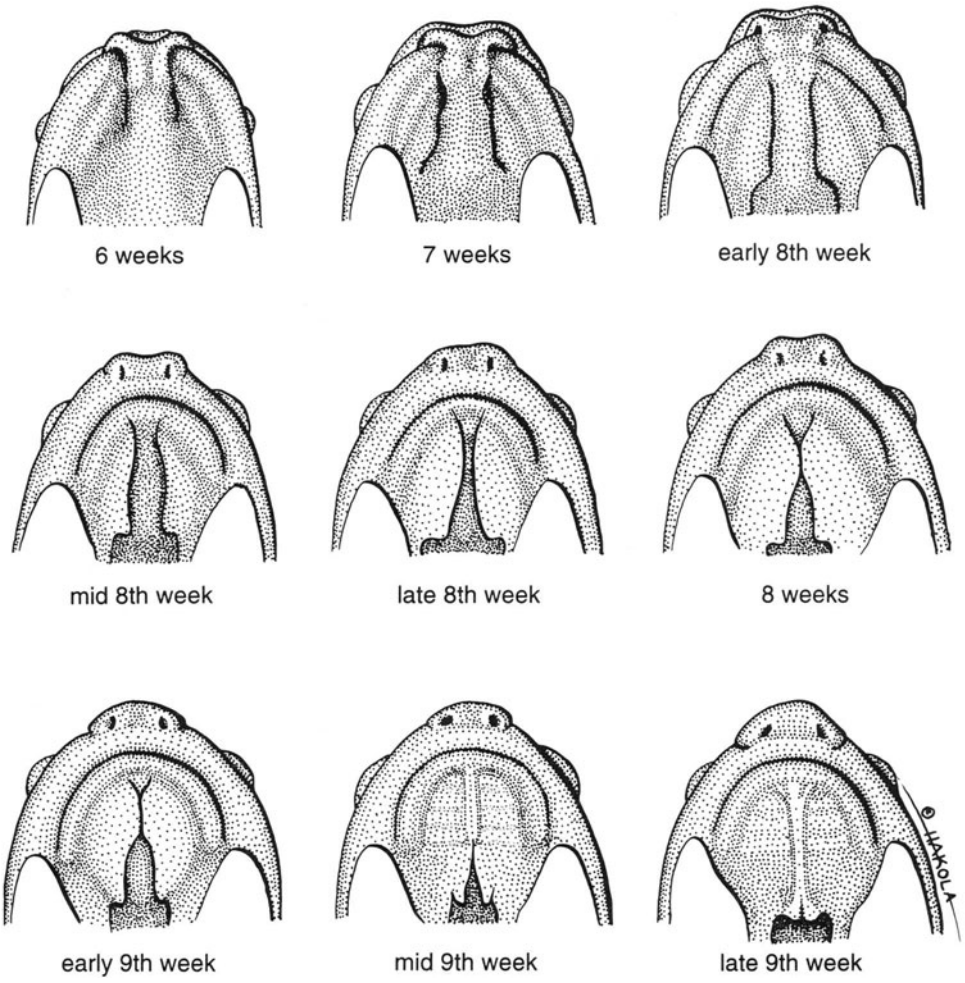

Figure 1.11. *Continued* (C) Development of the palate.

Development of Bone and Cartilage

Prior to discussing the actual development of the skull, it is important to describe briefly the development of bone and cartilage. Cartilage first appears in the embryo at approximately 5 weeks of age.[2] In the areas where cartilage will develop, the mesenchyme condenses while the cells proliferate and become rounded. Collagen and/or elastic fibers are then deposited in the intercellular matrix. The chondroblasts form the collagenous fibrils and the ground substance of the matrix.[2] Three types of cartilage (hyaline, fibrocartilage, and elastic) are distinguished according to the matrix formed.[2] Of these, hyaline cartilage is the most widely distributed.

Bone always develops by transformation of preexisting connective tissue.[1,2] It arises in two types of connective tissue, mesenchyme and cartilage. Like cartilage, bone consists of cells and an organic intercellular substance, the matrix. Intramembranous ossification is the type of bone formation that occurs in mesenchyme.[1,2] The mesenchyme first condenses and becomes highly vascular. Some cells differentiate into osteoblasts and begin to lay down osteoid. Calcium phosphate is then deposited into the osteoid tissue, which becomes organized into bone. Bone osteoblasts that we trapped in the matrix become osteocytes. At first, new bone has no organized pattern. The early spicules, however, become organized and coalesce into lamellae. Concentric lamellae, which form around blood vessels, are termed haversian systems. Between the surface plates of chordal bone, the intervening bone becomes spiculated or spongy.

Intracartilaginous or endochondral ossification is the type of bone formation that occurs in preexisting cartilaginous models.[2] In the long bone, a primary ossification center appears in the diathesis. The cartilage cells increase in size and the matrix becomes calcified as the cells die. Concurrently, a thin layer of bone is deposited under the perichondrium, becoming the periosteum. Invasion of vascular connective tissue from the periosteum breaks up the cartilage. Some of the invading cells differentiate into hemopoietic cells of the bone marrow, while others differentiate into osteoblasts, which deposit bone matrix on the spicules of the calcified cartilage. This process continues toward the epiphyses.[2]

Development of the Skull

The skull develops from mesenchyme surrounding the brain. It consists of neurocranium, a protective case from the brain, and viscerocranium, the main skeleton of the jaws.[1] A parachordal cartilage, or basal plate, forms around the cranial end of the notochord and fuses with the cartilage derived from the sclerotome regions of the occipital somites.[1] This cartilaginous mass eventually contributes to the base of the occipital bone.[1] The hypophyseal cartilage form around the developing pituitary gland and fuse to form the body of the sphenoid.[1]

Intramembranous ossification occurs in the mesenchyme at the sides and top of the brain forming the cranial vault, or calvaria. During fetal life, the flat bones of the vault are separated by dense connective tissue membranes or fibrous joints called sutures. The six large fibrous areas where several sutures meet are called fontanelles. The softness of the bones and their loose connections with the sutures enable the calvaria to undergo changes of shape or modeling during birth.[1] During this process the frontal bone becomes flat. The occipital bone becomes drawn out and one parietal bone slightly overrides or overlaps the other. Within a day or so after birth, the shape of the calvaria returns to normal.

The cartilaginous viscerocranium is that part of the cartilaginous skeleton from the first two pairs of branchial arches. After endochondral ossification, the dorsal end of the first arch cartilage (Meckel's cartilage) forms two middle ear bones, the malleus and incus. The dorsal end of the second arch cartilage (Reichert's cartilage) forms the stapes in the middle ear and the styloid process of the temporal bone.

Intramembranous ossification occurs within the maxillary prominence of the first branchial arch and subsequently forms the maxilla, zygoma, and squamous temporal bones. The squamous temporal bone later becomes incorporated into the neurocranium.[1] The mesenchyme of the mandibular prominence condenses around the first arch cartilage and undergoes intramembranous ossification to form the mandible. Some endochondral ossification occurs at the center of the chin and in the mandibular condyle.

In summary, the earliest indication of the skull is a massive dense mesenchyme, which during the fifth and sixth weeks envelopes the cranial end of the notochord and extends into the nasal region. Laterally, it expands into wings that are continuous with the general head mesoderm that houses the brain. Eventually, it communicates within the mesenchymal cores of the branchial arches.

During the seventh week, chondrification begins medially in the future occipital and sphenoidal regions of the blastemal skull. Chondrification spreads outward, upward, and into the nose. The internal ears become invested with cartilaginous otic capsules, which eventually unite with the occipital and sphenoidal cartilage. The chondrocranium (Fig. 1.12A and 1.12B), as it is termed, is thus confined chiefly to the base of the skull, whereas the rest of the sides and roof of the calvarium are represented by a connective tissue capsule in which the membranous bones are destined to appear. Chondrification also occurs more or less extensively in the branchial arches. The process as a whole reaches its height by the

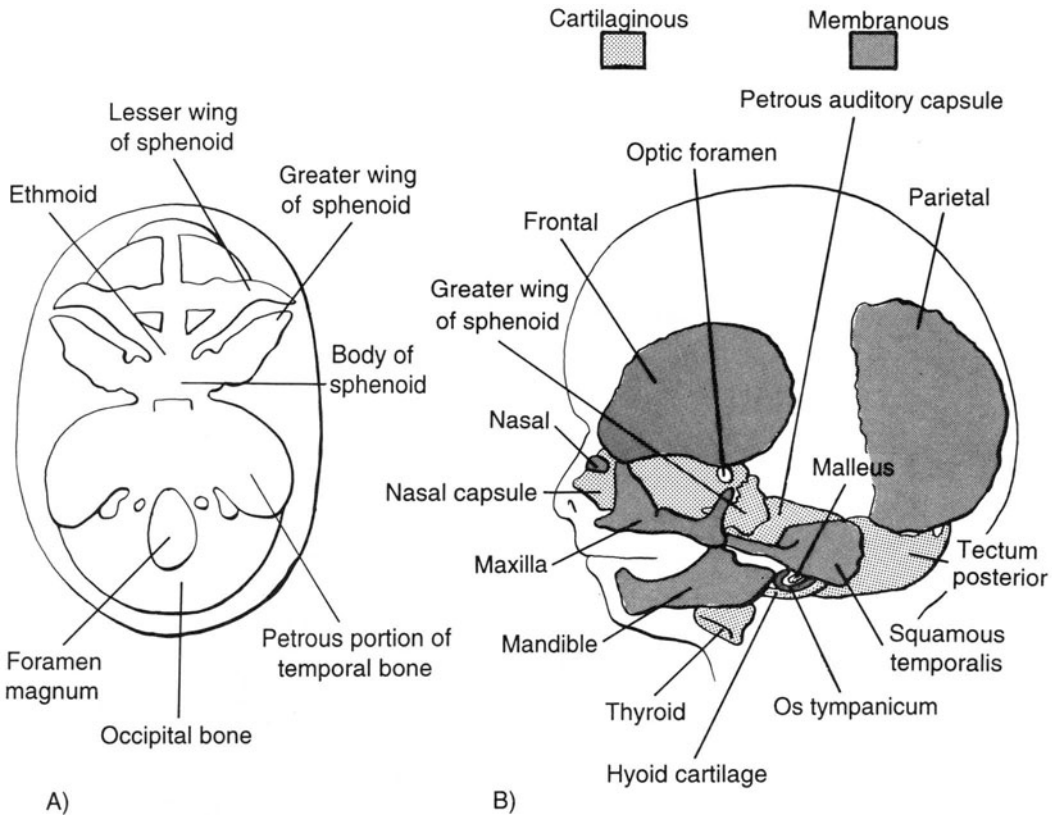

Figure 1.12. (A) The cartilaginous neurocranium or chondrocranium at 12 weeks (endochondral bone formation). (B) The derivation of bones of the fetal skull at 20 weeks; both endochondral and membranous bone formation.

middle of the third month, when the chondrocranium is a unified cartilaginous mass without clear boundaries.[2]

During the period of ossification that now ensures, it appears that the majority of bones develop from two or more formative centers. The basal part of the skull is modeled in cartilage. Endochondral ossification gradually occurs, replacing this cartilaginous base. By contrast, the bony sides and roof of the skull develop directly in the membrane of connective tissue.

Skull Malformations

Abnormalities may range from major defects that are incompatible with life to those are minor and insignificant.

Acrania

The cranial vault is virtually absent. An extensive defect of vertebral column is frequently present. Acrania-associated anencephaly occurs about once in 1000 births and is incompatible with life. The malformation results from failure of the cranial end of the neural tube to close during the fourth week and subsequent failure of the cranial vault to form.[1]

Craniosynostosis

A variety of calvarial deformities result from premature closure of the skull sutures. Prenatal closure results in the most severe abnormalities. The cause of craniosynostosis is frequently unknown, but genetic factors are important influences, especially in those associated with syndromes. The type of skull deformation depends upon which sutures close prematurely. When the sagittal suture prematurely fuses, the calvarium becomes long and narrow and has a scaphocephalic appearance. If a coronal or lambdoid suture closes prematurely on one side only, the calvarium adapts by compensatory overgrowth, leading to typical plagiocephalic deformities.

Microcephaly

Infants with microcephaly are born with a slightly small cranium. Fontanelles close early during infancy and the sutures close during the first year. Microcephaly is not caused by premature closure of the sutures, but rather from an abnormality of the central nervous system in which the brain fails to expand, pushing the skull vault outward.[1] Generally, microcephalics are neurologically compromised.

Development of the Brain

The cranial four pair of somites of the neural tube develop into the brain. In the fourth week of gestation, fusion of the neural folds in the cranial region and closure of the rostral neuropore results in the formation of three primary brain vesicles.[1] These three primary brain vesicles form (1) the forebrain, or prosencephalon; (2) the midbrain, or mesencephalon; and (3) the hindbrain, or rhombencephalon.[1] During the fifth week, the forebrain further divides into two vesicles, the telencephalon and the diencephalon, and the hindbrain partially divides into the metencephalon and the myelencephalon.[1] As a result, there are now five secondary brain vesicles. These go on to form the definitive adult brain (Fig. 1.13).

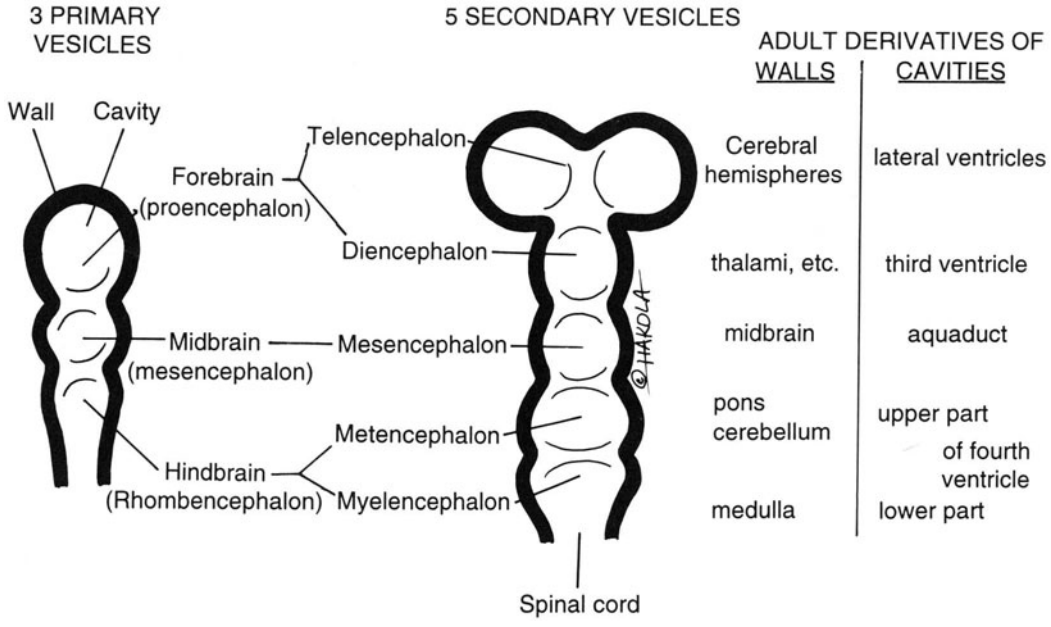

FIGURE 1.13. Brain vesicles and their adult derivatives.

The Forebrain (Prosencephalon)

Before closure of the rostral neuropore, two lateral outgrowths or diverticula, called optic vesicles appear, one on each side of the forebrain (Fig. 1.14). The optic vesicles are the primordia of the retina of the eye and optic nerves. A second pair of diverticula soon arise more dorsally. These are called the telencephalic vesicles, which are the primordia of the cerebral hemispheres, their cavities becoming the lateral ventricles.

The Pituitary Gland

The pituitary gland is interesting in that it develops from two different sources, an upgrowth of the ectoderm of the stomadeum and a downgrowth from the neuroectoderm of the diencephalon[1] (Fig. 1.15). This double origin explains why the pituitary gland is composed of two completely different types of tissue. The adenohypophysis (glandular portion) arises from the oral ectoderm and the neurohypophysis (nervous portion) originates from the neuroectoderm.[1] At approximately 24 days, a diverticulum called Rathke's pouch arises from the roof of the stomadeum and grows upward toward the brain. By the fifth week, this pouch has elongated and become constricted at its attachment to the oral epithelium, giving it a nipple-like appearance. At this stage, it comes into contact with the infundibulum, a ventral downgrowth of the diencephalon (Fig. 1.15). The stalk of Rathke's pouch passes between the chondrification centers of the developing presphenoid and dasysphenoid bones of the skull. During the sixth week, the connection of Rathke's pouch with the oral cavity disappears. A remnant of the stalk may persist, giving rise to a pharyngeal hypophysis in the pharyngeal roof.[1]

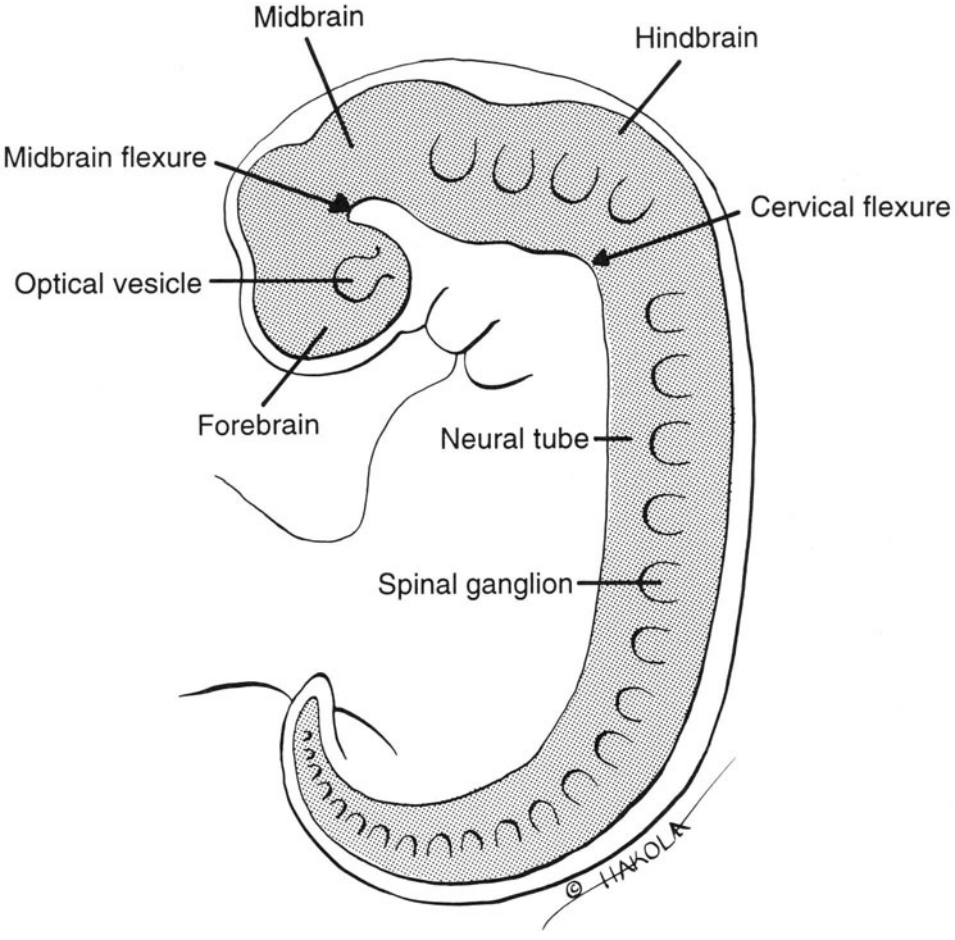

Figure 1.14. Schematic lateral view of embryo showing flexures demarcating the primary divisions of the brain. Note the optic vesicle.

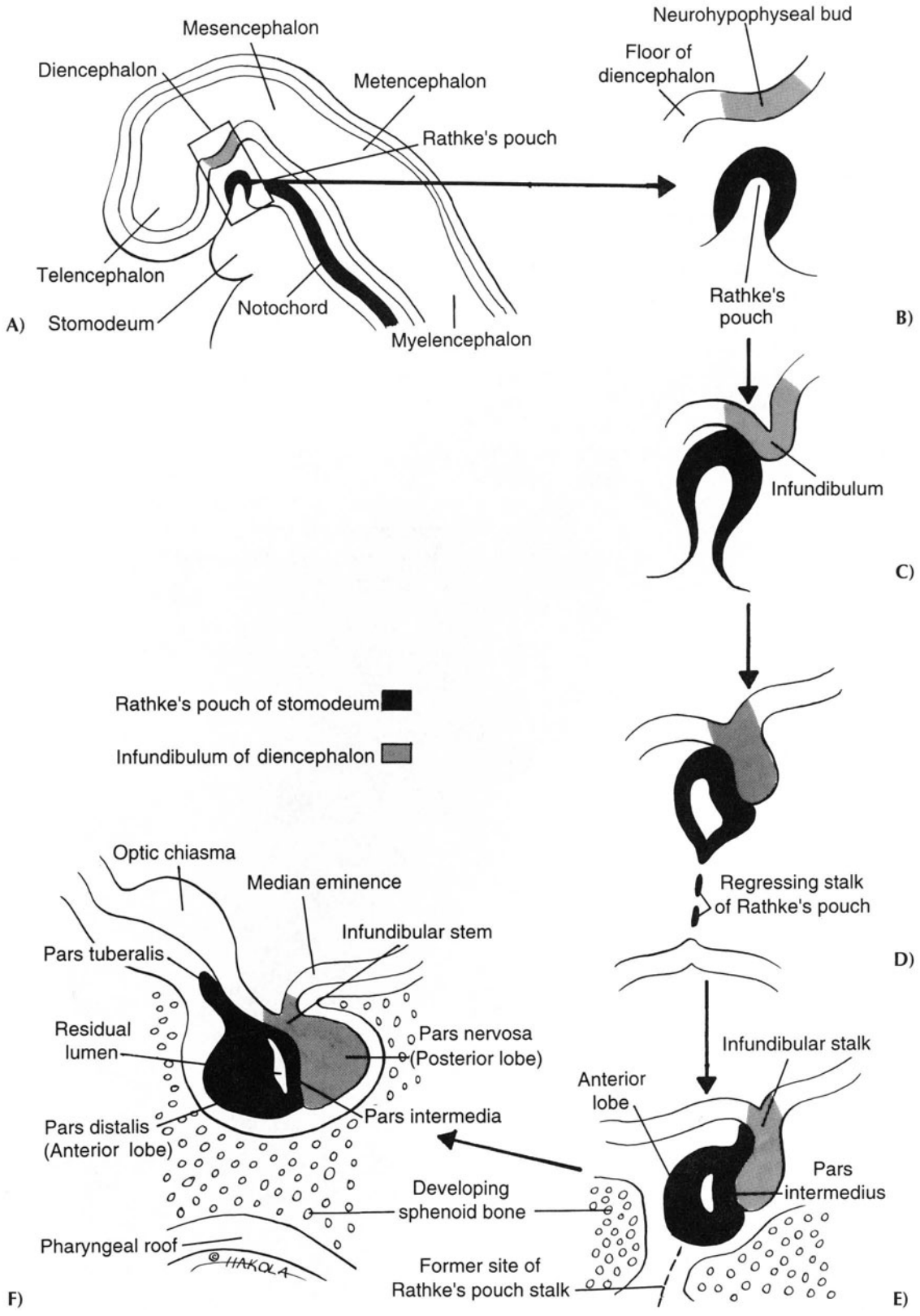

Figure 1.15. Diagrams illustrating development of the pituitary gland. (A) Sagittal section of 24-day embryo showing Rathke's pouch as an upgrowth from the stomodeum and the neurohypophyseal bud from the hindbrain. (B–D) Successive stages of the developing pituitary gland. By 8 weeks, Rathke's pouch loses its connection with the oral cavity. (E, F) Later stages showing anterior wall of Rathke's pouch forming the anterior lobe of the pituitary (adenohypophysis).

Congenital Malformations of the Brain

Abnormal development of the brain is not uncommon, presumably because of the complexity of its embryonic origin.[1] Most major congenital malformations of the brain and overlying tissues result from defective closure of the rostral neuropore during the fourth week.[1] Defects in the formation of the cranium (cranium bifidum) are often associated with malformations of the brain and/or meninges.[1] Such defects are often in the median plane and usually involve the cranial vault.[1] When the defects in the cranium are small, only the meninges themselves herniate and the malformation is called a meningocele. When the cranial defect is large, however, the meninges and part of the brain form a meningoencephocele.

Severe malformations of the brain result when the rostral neuropore fails to close.[1] As a result, the forebrain premordium is abnormal and the development of the calvaria is severely defective. The brain is exposed or extrudes from the skull, a condition termed exencephaly.

Owing to the abnormal structure and vascularization of the embryonic exencephalic brain, the nervous tissue degenerates and is replaced by a vascular mass consisting mostly of hindbrain structures.[1] This condition is called anencephaly. Fortunately, sustained extrauterine life is impossible in infants born with anencephaly.

Rhinencephalic Dysplasia

This condition is characterized by forebrain malformations and underdevelopment of the midline structures of the face (Fig. 1.16). The cerebral anomalies present because of failure of the procencephalon to divide either transversely into the telecephalon and diencephalon, or sagittally into the cerebral hemispheres.[1] The facial defects include varying degrees of hypotelorbitism, and absence or severe hypoplasia of the nose and premaxillary.

Starting with cyclopia, which represents the most severe example, the spectrum of faciocerebral anomalies with hypotelorbitism moves through ethmocephaly, cebocephaly, and median cleft lip without a premaxilla, toward normal.

Figure 1.16. A clinical example of holoprosencephaly.

Specialized Structures of the Face

The Eye and Ear

The visual organs, or eyes, develop from three sources: (1) neuroectoderm of the forebrain, (2) surface ectoderm of the head, and (3) mesoderm between the aforementioned layers.[1] Eye formation is first evident at approximately 22 days of fetal life.[1] It is at this time that a pair of grooves called optic sulci appear in the neural folds at the cranial end of the embryo. As the neural folds fuse to form the prosencephalon, or forebrain, the optic sulci invaginate to form a pair of hollow diverticula called optic vesicles, which project from the sides of the forebrain into the adjacent mesenchyme (Fig. 1.17). The formation of the optic vesicles is induced by the mesenchyme adjacent to the developing brain.

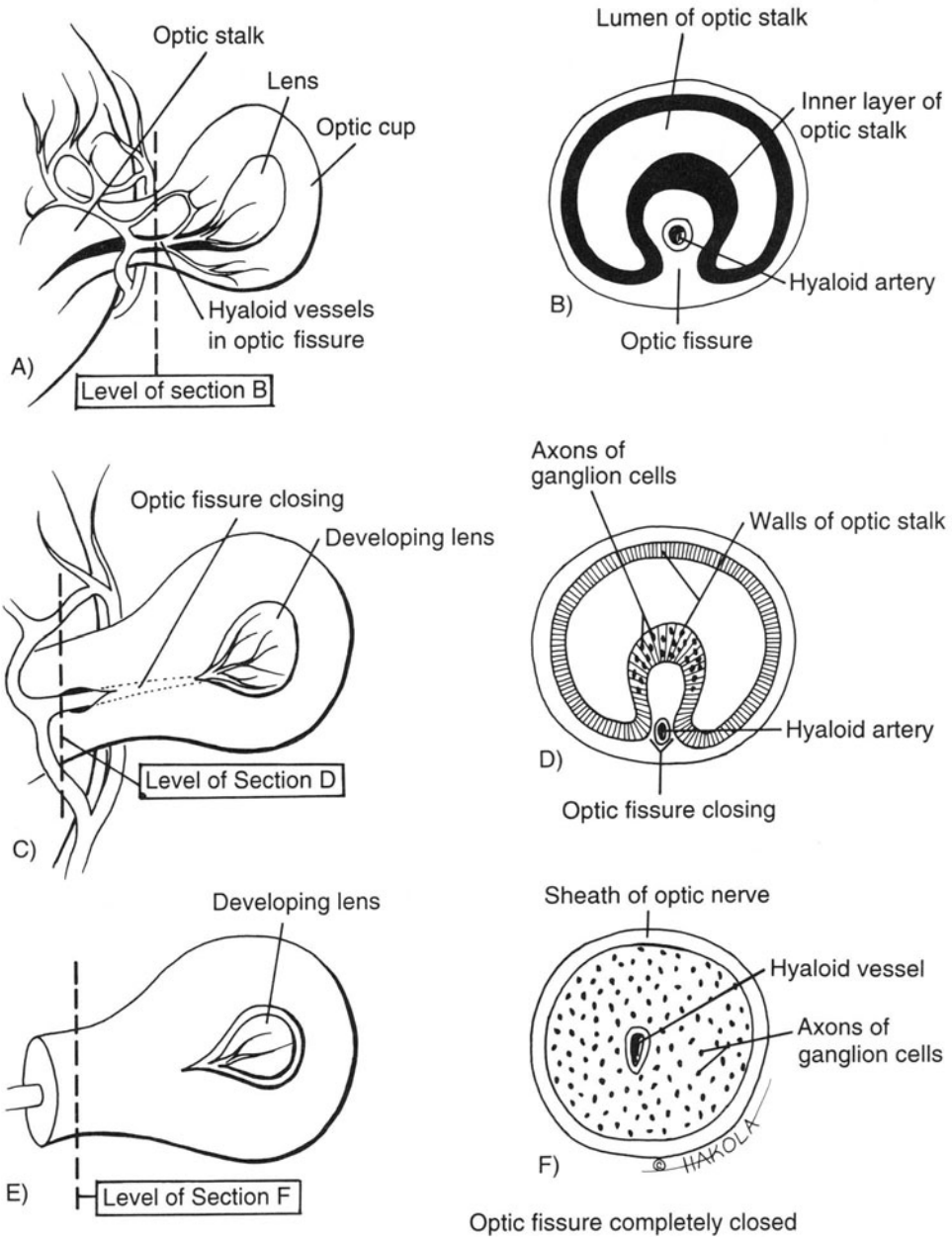

Figure 1.17. Diagrams illustrating changes in the optic stalk, closure of the optic fissure, and formation of the optic nerve. (These changes occur between the sixth and eighth weeks of intrauterine development.)

As the bulb-like optic vesicles grow laterally, their distal ends expand and their connections with the forebrain constrict to form optic stalks (Fig. 1.17). As the optic vesicles grow outward, displacing the mesenchyme, their external surface becomes flattened.

Concurrently, the surface ectoderm adjacent to the optic vesicles thickens to form the lens placodes (Fig. 1.17). The central region of each lens placoded soon invaginates and sinks below the surface, forming a lens pit (*fovea lentis*). The edges of this pit gradually approach each other and fuse to form a spherical lens vesicle, which becomes pinched off from the surface ectoderm. It is totally surrounded by mesenchyme. The lens vesicle develops into the lens of the eye (Fig. 1.17).

As the lens vesicles are developing, the optic vesicles invaginate and become double-walled, cup-like structures called optic cups (Fig. 1.18). Linear grooves called choroid fissures, or optic fissures, develop on the inferior surface of the optic cups and along the optic stalks. Hyaloid blood vessels develop in the mesenchyme in these fissures. The hyaloid artery, a branch of the ophthalmic artery, supplies the inner layer of the optic cup, the lens vesicle, and the mesenchyme in the optic cup. The hyaloid vein returns blood from these structures. As the edges of the optic fissure come together and fuse, the hyaloid vessels are enclosed within the optic nerve. The distal portions of the hyaloid vessels eventually degenerate, but their proximal portions persist as the central artery and vein of the retina. The retina develops from the walls of the optic cup, which is an outgrowth of the brain (diencephalon). The pigmented portion of the epithelium of the ciliary body is derived from the outer layer of the optic cup, and it is continuous with the pigment epithelium of the retina. The nonpigmented portion of the ciliary epithelium represents the forward prolongation of the neural layer of the retina, in which no neural elements differentiate. The iris develops from the edge of the optic cup, which bends inward and partially covers the lens (Fig. 1.18). The lens develops from the hollow lens vesicle, a derivative of the surface ectoderm. The anterior chamber develops from a cleft-like space that forms in the mesenchyme located between the developing lens and the surface ectoderm. The mesenchyme superficial to this space forms the substantia propria of the cornea and the mesothelium of the anterior chamber. The posterior chamber develops from a space that forms in the mesenchyme posterior to the developing iris and anterior to the developing lens. The mesenchyme surrounding the optic cup differentiates into an inner vascular layer, the choroid, and an outer fibrous layer, the sclera.

The eyelids develop from two ectodermal, or cutaneous, folds containing cores of mesenchyme. The eyelids meet and adhere by about the 10th week and remain adherent until the 26th week. While the eyelids are adherent, the closed conjunctival sac exists anterior to the cornea; when the eyes open, the conjunctiva covers the white of the eye and lines the eyelids. The muscles and tarsal plates develop from the mesenchyme and the cores of the eyelids. It is generally stated that the upper and lower eyelids are separated by 26 weeks. The size of the eyes of the newborn infant is about two-thirds that of the eyes of an adult. The eyes grow fairly rapidly in the first year and then grow more slowly until puberty; after this, additional growth is negligible.

Congenital Malformations of the Eye

Because of the complexity of eye development, many abnormalities may occur, but most of them are relatively rare.

Congenital Colobomas

Colobomas are seen in the lower eyelid and are associated with Treacher Collin's syndrome. Palpebral colobomas probably result from a local developmental disturbance in the growth of the eyelid.

Congenital Glaucoma

High intraocular pressure and enlargement of the eye result from abnormal development of the drainage mechanism of the aqueous humor. Congenital glaucoma is usually caused by recessive mutant genes, but the condition sometimes results from maternal rubella infection during early pregnancy.

Congenital Cataract

In this condition, the lens is opaque and frequently appears grayish-white. Many lens opacities are inherited, but some are caused by noxious agents, particularly rubella virus.

Congenital Ptosis

Drooping of one or both upper eyelids at birth is relatively common. It results from abnormal development or failure of development of the levator palpebrae superioris muscle. Congenital ptosis may also result from incomplete innervation of this muscle.

Microphthalmos

The eye on one side may be very small and may be associated with gross ocular abnormalities, or it may be a normal-appearing miniature eye. The affected side of the face is underdeveloped and the orbit is small. Microphthalmos may be associated with other congenital abnormalities, such as facial clefts, or may be part of a syndrome, Trisomy 13. Microphthalmos results from arrested development of the eye after the optic vesicle is formed. If the interference with development occurs be-

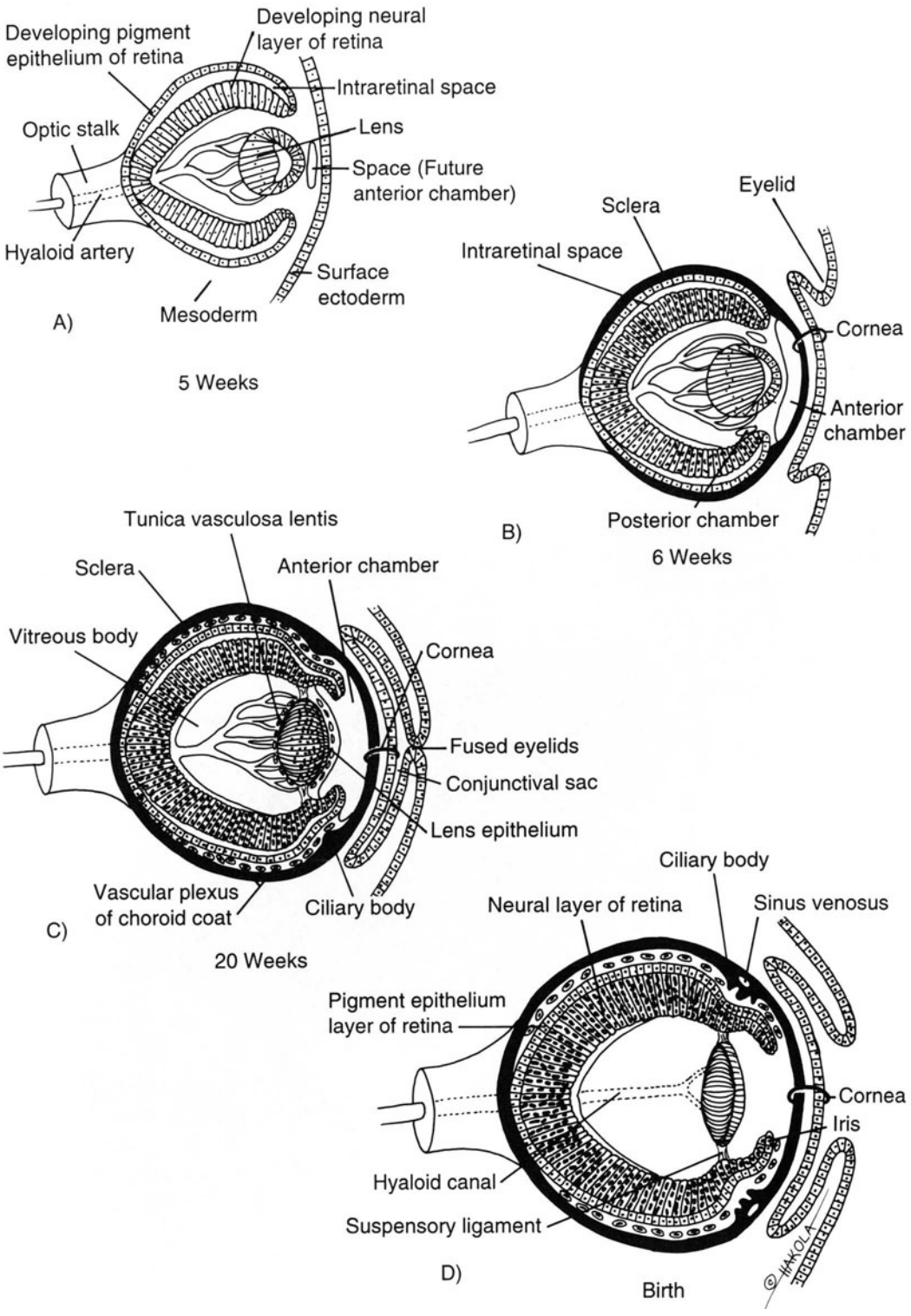

Figure 1.18. Diagrams illustrating the development of the retina and the optic nerve from the optic cup and optic stalk. (From 5 weeks intrauterine to birth.)

fore the embryonic optic fissure closes, microphthalmos is associated with gross ocular defects. When eye development is arrested later in the embryonic or fetal periods, simple microphthalmos results (small eye without gross ocular abnormalities). Most cases of so-called simple microphthalmia are probably caused by infectious agents that cross the placental membrane during the fetal period.

Anophthalmos

In this condition, the eyelid forms, but no eyeball is present. In some cases, eye tissue may be recognizable histologically. Anophthalmia may be unilateral or bilateral. Usually, there are no other defects. In primary anophthalmos, eye development is arrested early and results from failure of the optic vesicle to form. In secondary anophthalmos, the entire forebrain is suppressed and absence of the eye is one of several malformations. In consecutive anophthalmos or degenerative anophthalmos, the optic vesicle develops and then undergoes degeneration, probably as a result of a teratogenic agent.

Cyclopia

In this very rare condition, the eyes are partially or completely fused into a single median eye enclosed in a single orbit. Usually there is a proboscis above the eye. Cyclopia appears to result from suppression of midline cerebral structures that develop from the cranial part of the neural plate.

Ears

The ear consists of three anatomical parts: external, middle, and internal. The external and middle parts are concerned mainly with the transference of sound waves from the exterior to the internal ear, which contains the vestibulocochlear organ concerned with equilibration and hearing.

Internal Ear

The internal ear (Fig. 1.19A and 1.19B) is the first of the three anatomical divisions of the ear to appear. Early in the fourth week, a thickened plate of surface ectoderm,

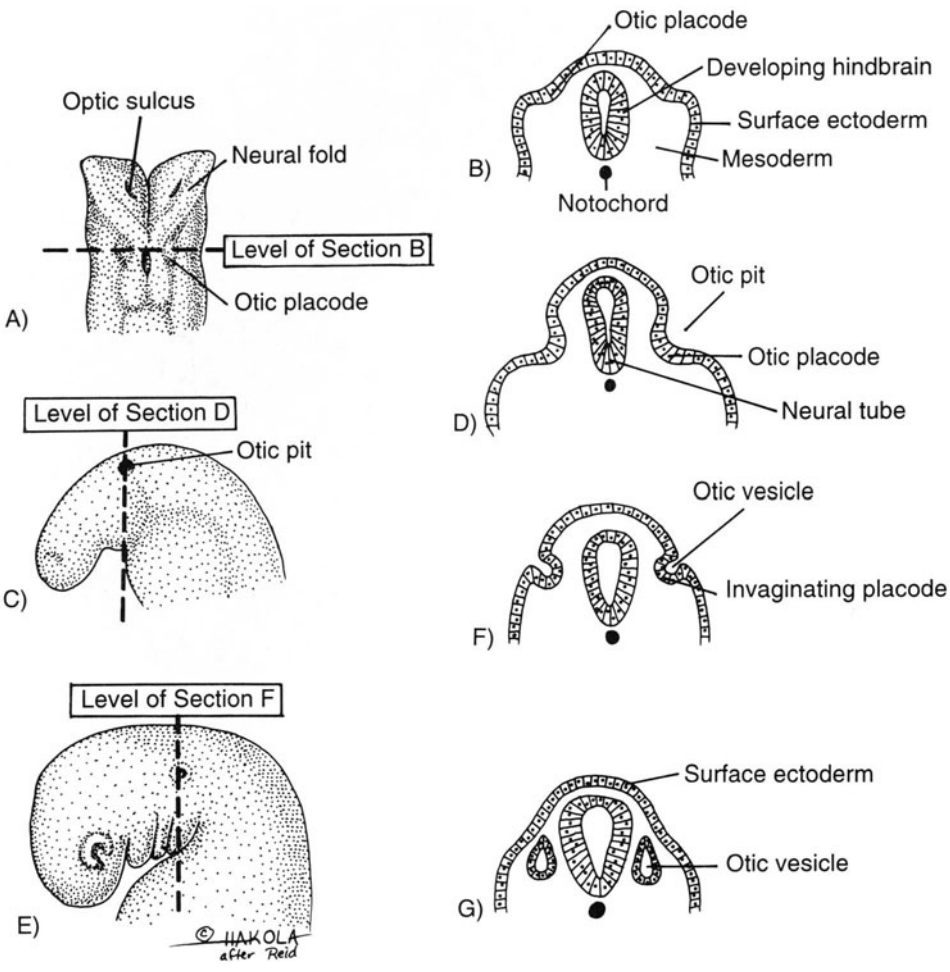

Figure 1.19. (A) Diagrams illustrating development of the inner ear. Starting first with the otic pit to the progressive development of the otic vesicle. (From 22 to 28 days intrauterine.)

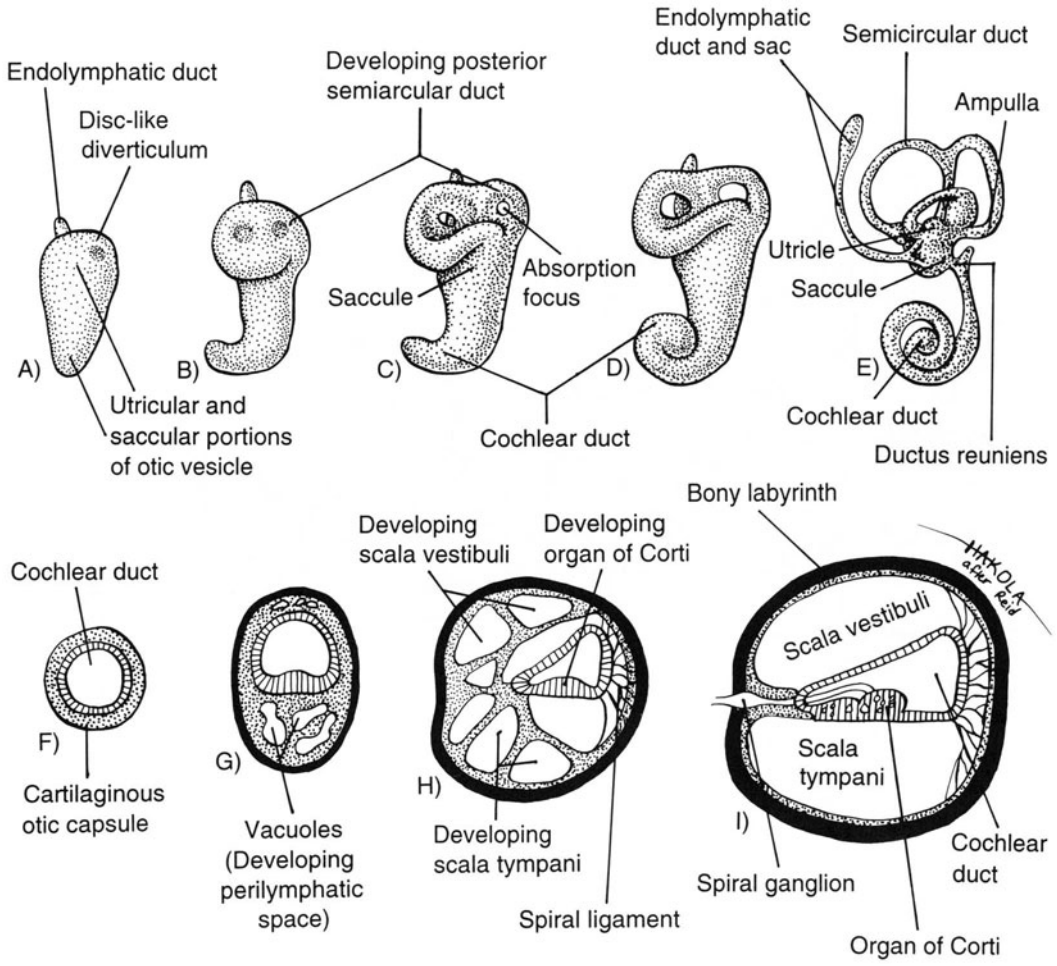

Figure 1.19. *Continued* (B) Diagrams illustrating development of the inner ear. This shows the development of the otic vesicles into the membranous labyrinth, semicircular canals, cochlear duct, organ of Corti, and perilymphatic spaces. (From 5th to 20th week intrauterine.)

the otic placode, appears on each side of the developing hindbrain. Each placode soon invaginates and sinks below the surface ectoderm into the underlying mesenchyme to form an otic pit. The edges of the pit come together and fuse to form an otic vesicle (otosis), the primordium of the membranous labyrinth. The otic vesicle soon loses its connection with the surface ectoderm. A hollow diverticulum grows out from the otic vesicle and elongates to form the endolymphatic duct and sac.

Two regions of each optic vesicle soon become recognizable: a dorsal utricular portion, from which the utricle, semicircular ducts, and endolymphatic duct arise; and a ventral saccular portion, which gives rise to the saccule and the cochlear duct. From the ventral saccular portion of the otosis, a tubular diverticulum, the cochlear duct, grows and coils to form the cochlea. The connection of the cochlea with the saccule becomes constricted to form the narrow ductus reunions.

The organ of Corti differentiates from cells in the wall of the cochlear duct. Ganglion cells of the VIIIth cranial nerve migrate along the coils of the cochlea and form the cochlear (spiral) ganglion, from which nerve processes grow to the organ of Corti, where they terminate on the hair cells.

The Middle Ear

Figure 1.20 illustrates the development of the middle ear. The distal portion of the first pharyngeal pouch expands and becomes the tympanic cavity. The proximal unexpanded portion becomes the auditory tube. As the tympanic cavity expands, its endodermal epithelium gradually envelopes the middle ear bones, or auditory ossicles (malleus, incus, and stapes), their tendons and ligaments, and the chordae tympani nerve. All these structures receive more or less complete epithelial investment. During the late fetal period, the expansion of the tympanic cavity gives rise to the mastoid antrum.

The External Ear

The external acoustic meatus (Fig. 1.21) develops from the dorsal end of the first branchial groove. The ectoder-

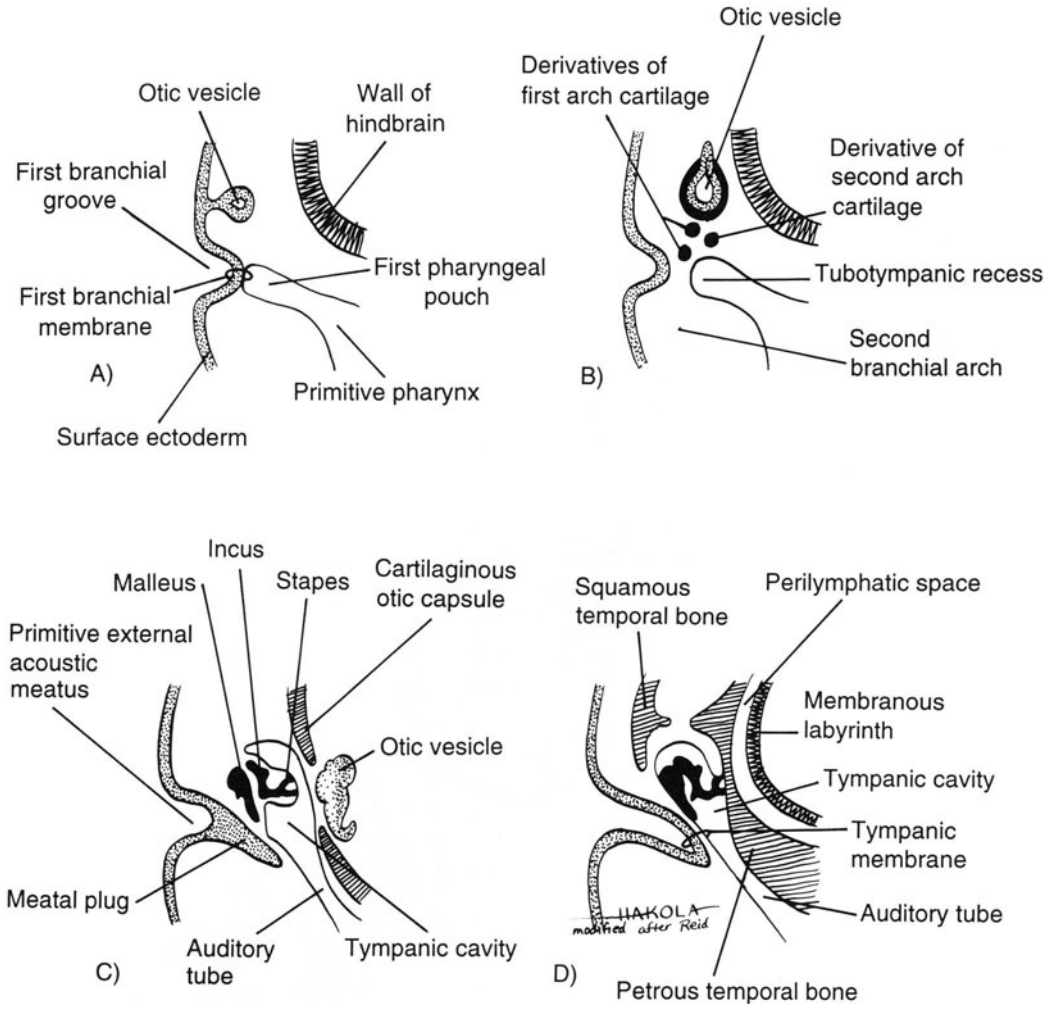

Figure 1.20. Diagrams illustrating development of the middle ear. From the first branchial pouch and the first and second branchial arches come the tympanic cavity and the ossicles of the ear.

mal cells at the bottom of this funnel-shaped tube proliferate and extend inward as a solid epithelial plate called the meatal plug. Late in the fetal period, the central cells of this plug degenerate, forming a cavity that becomes the inner part of the external acoustic meatus. The early tympanic membrane is represented by the first branchial membrane, which separates the first branchial groove and the first pharyngeal pouch. As development proceeds, mesenchyme grows between the branchial membrane and later differentiates into the fibrous stratum of the tympanic membrane. Thus, the tympanic membrane develops from the ectoderm of the meatal plug, the endoderm of the tubotympanic recess, and the mesenchyme of the first and second branchial arches.

The auricle develops from six swellings called auricular hillocks, which arise around the margins of the first branchial groove. These swellings are produced by proliferation of mesenchyme from the first and second branchial arches. As the auricle grows, the contribution of the first branchial arch becomes relatively reduced. The lobule is the last part of the auricle to develop. The external ears begin to develop in the upper part of the future neck region, but as the mandible develops, the auricles move to the side of the head and ascend to the levels of the eyes.

Congenital Malformations of the Ear

Congenital Deafness

Congenital impairment of hearing may be the result of maldevelopment of the sound-conducting apparatus of the middle ear or of the neurosensory (perceptive) structures of the inner ear. Most types of congenital deafness are caused by genetic factors. Rubella infection during the critical period of embryonic development of the ear (particularly during the seventh and eighth weeks) can also cause maldevelopment of the organ of Corti. Congenital deafness may be associated with maternal goiter, which may result in fetal hypothyroidism. Congenital fixation of the stapes results in severe conductive deafness in an otherwise normal ear. Defects of the malleus

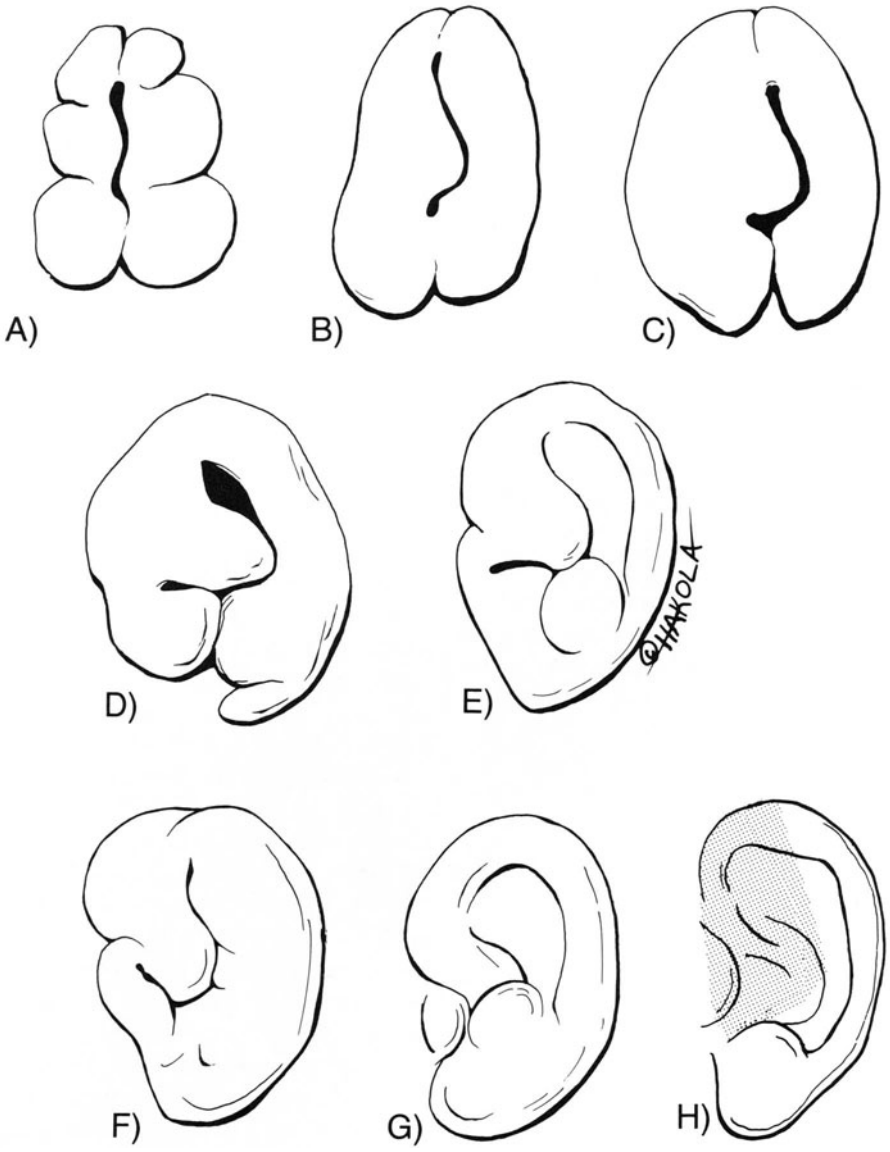

Figure 1.21. Diagrams showing the development of the external ear auricle from the six auricular hillocks. (From 6th to 32nd intrauterine week.) (H) Represents the adult fully formed ear with remnants of Arches I, II, III shaded and IV, V, VI nonshaded.

and incus are often associated with the first arch syndrome.

Auricular Abnormalities

Auricular appendages or tags are relatively common and result from the development of accessory auricular hillocks. They usually appear anterior to the auricle, more often unilateral than bilateral. Absence and hypoplasia of the auricle are rare and are usually associated with the first arch syndrome. Anotia results from failure of the auricular hillock to develop, and microtia results from suppressed development of the auricular hillock. Atresia of the external acoustic meatus and middle ear abnormalities are usually also present.

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CHAPTER 2

Craniofacial Growth

Steven R. Cohen and Andrew Wexler

Significance

Growth is the essence of the developing human being.[1] Physical growth starts shortly after fertilization and continues through life. Growth of different parts of the body follows predictable schedules during normal development and maturation. This timetable of development is influenced and controlled by many genetic and environmental factors. Any disturbance in normal development and growth may lead to a disproportion of physical features. Imbalances may be transient and sometimes compensated for by later catch-up growth. Most syndromes with dysmorphic features, however, display more or less recognizable disturbances in growth and development.[1]

The face is unique both because of its variability in size and shape, as well as its expression of personal emotion. Evaluation of the craniofacial region is therefore dynamic and complex. Like the rest of the body, the ultimate shape of the human head and face depends on a variety of interactive genetic and environmental forces. Intrinsic and extrinsic pressure and neuromuscular function contribute to the overall shape. With growth and development the infant's face gradually undergoes marked changes. The chin develops, the jaws enlarge both horizontally and vertically, and the eyes appear less wide set (Fig. 2.1). The purpose of this chapter is to summarize how facial growth and development is thought to occur. Growth of specific craniofacial regions, such as the orbitocranial, midface, and mandible, will be discussed. Clinical situations where growth has gone awry will be presented.

Theories of Facial Growth

Growth is not merely a process of increasing size. Rather, progressive facial enlargement is a differential growth process in which each of the many component parts matures at a different rate than others, and to a different extent, in a myriad of directions.[2] It is a gradual maturational process involving different, but functionally interrelated tissues and organs. The growth process involves a succession of regional changes in proportions, requiring countless localized adjustments to achieve ultimate function among the parts. The child's face is not merely a miniature of the adult's (Fig. 2.1).

An indepth understanding of facial morphogenesis is essential so the clinician can properly grasp: (1) the differences between normal and the ranges of abnormal; (2) biologic reasons for these differences and the unlimited variations; (3) reasons for rationales used in diagnosis, treatment planning, and selection of clinical procedures; and (4) the biologic factors underlying important clinical problems of growth and relapse after treatment.[2]

The addition of new bone on one side of the bony cortex and its loss from the other allows skeletal growth to proceed (Fig. 2.2). The surface facing the direction of growth is depository whereas the surface facing away is resorptive. This composite process is termed *drift*, which provides the mechanism of direct growth movement of any given area of bone.[2]

The outside and inside surfaces of bone are completely blanketed by a mosaic-like pattern of growth fields[3] (Fig. 2.2). The combinations of depository and resorptive fields produce characteristic growth movements. These resorptive and depository growth fields do not have the same rate of activity.[2] Some depository fields grow more rapidly and to a greater extent than others. The same is true for resorptive fields. Fields that have special significance in the growth process have been called *growth sites*.[2] The mandibular condyle, for example, is such a growth site.

Remodeling is basic to the growth process.[2] Sequential remodeling in shape and size of each region must occur. This progressive, sequential movement of component parts as the bone enlarges is termed *relocation* (Fig. 2.3).[2]

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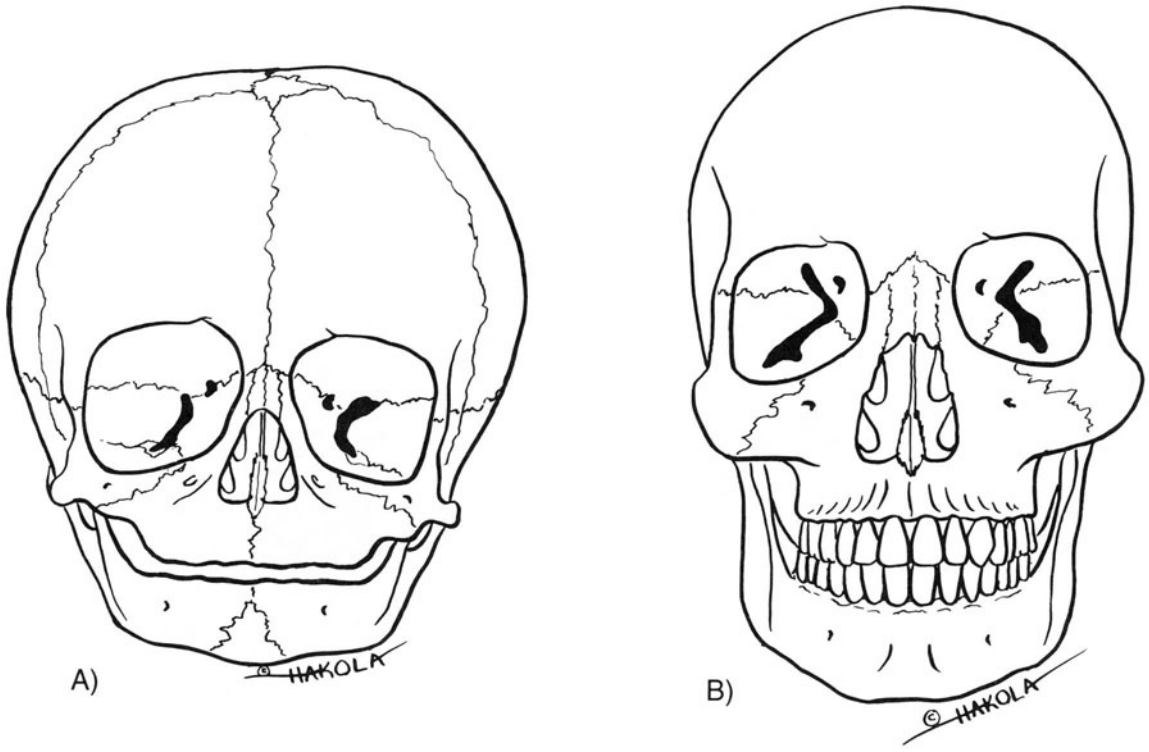

Figure 2.1. Skeletal changes that occur from infancy (A) to adulthood (B).

As bone enlarges, it is simultaneously carried away from adjacent bones. This process is termed *primary displacement*.[2] A process of *secondary displacement* also occurs during growth.[2] Primary displacement is associated with a specific bone enlargement. Secondary displacement, however, is the movement of a whole bone caused by separate enlargement of other adjacent or distant bones. For example, increases in the size of bones that compose the middle cranial fossa result in marked displacement of the maxillary complex anteriorly and inferiorly (Fig. 2.4).

The process of facial growth requires an intimate relationship between its soft and hard tissue components. One thus realizes that no part is truly independent and self-contained.[2] Continuous remodeling is involved in individual bones, as well as in adjacent and distant bones, as changes in the relative position of individual bones occur.

Control of Facial Growth

Central to any discussion on growth control is the question of *genetic influences*.[2] Genetic preprogramming has been presumed to be a "fundamental influence in establishing basic facial patterns and features."[2] It has yet to be demonstrated, however, that genes are the exclusive determinants for all growth parameters, including regional growth amounts, velocities, and the minute details of regional configuration.[2] Although genes are a basic participant in a given cell's function, the mechanisms by which extracellular conditions activate an intracellular process are important.

Historically, *biomechanical forces* acting on bone were thought to have an important role in regulating its development, morphologic configuration, histologic structure, and physical properties. *Wolfe's law* of bone transformation, introduced in 1899, is still a useful working concept.[2] Basically, Wolfe felt that bone grows and develops in a manner that is a composite of the physiologic forces exerted on it. Thus, the ultimate structure of bones is adapted to its function. Biomechanical forces play an important role in the bony developmental process; however, it is likely to be a secondary role.[2] It is thought that such physical forces are involved in activation of osteogenetic connective tissues.

Earlier in the twentieth century, it was thought that the growth, form, and dimensions of a specific bone were governed by intrinsic programming residing within the cells of periosteum, sutures, and bone-related cartilages.[2] Although influences such as hormones and muscle

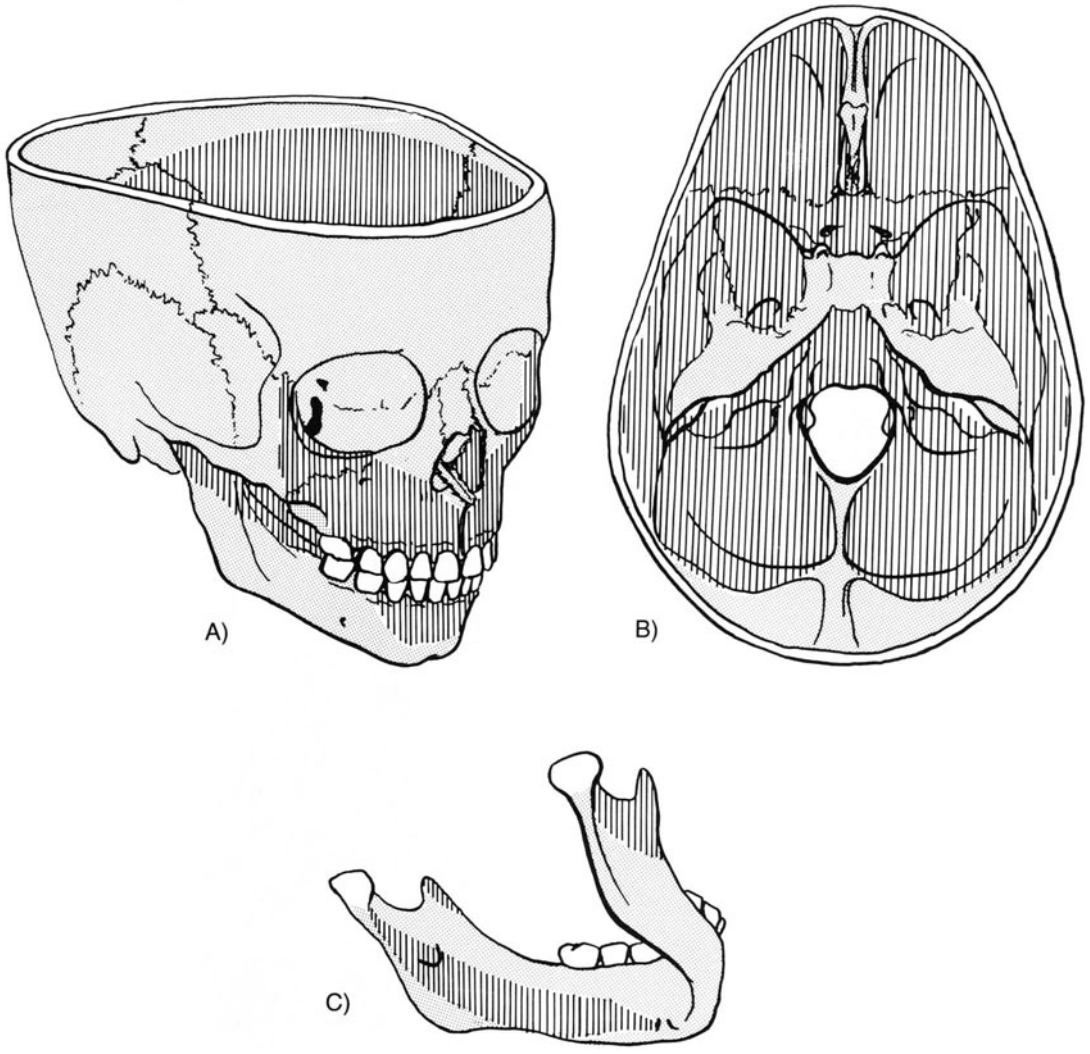

Figure 2.2. Resorption and depository fields of growth (A, B, C). Striped areas are resorptive; stippled areas are depository.

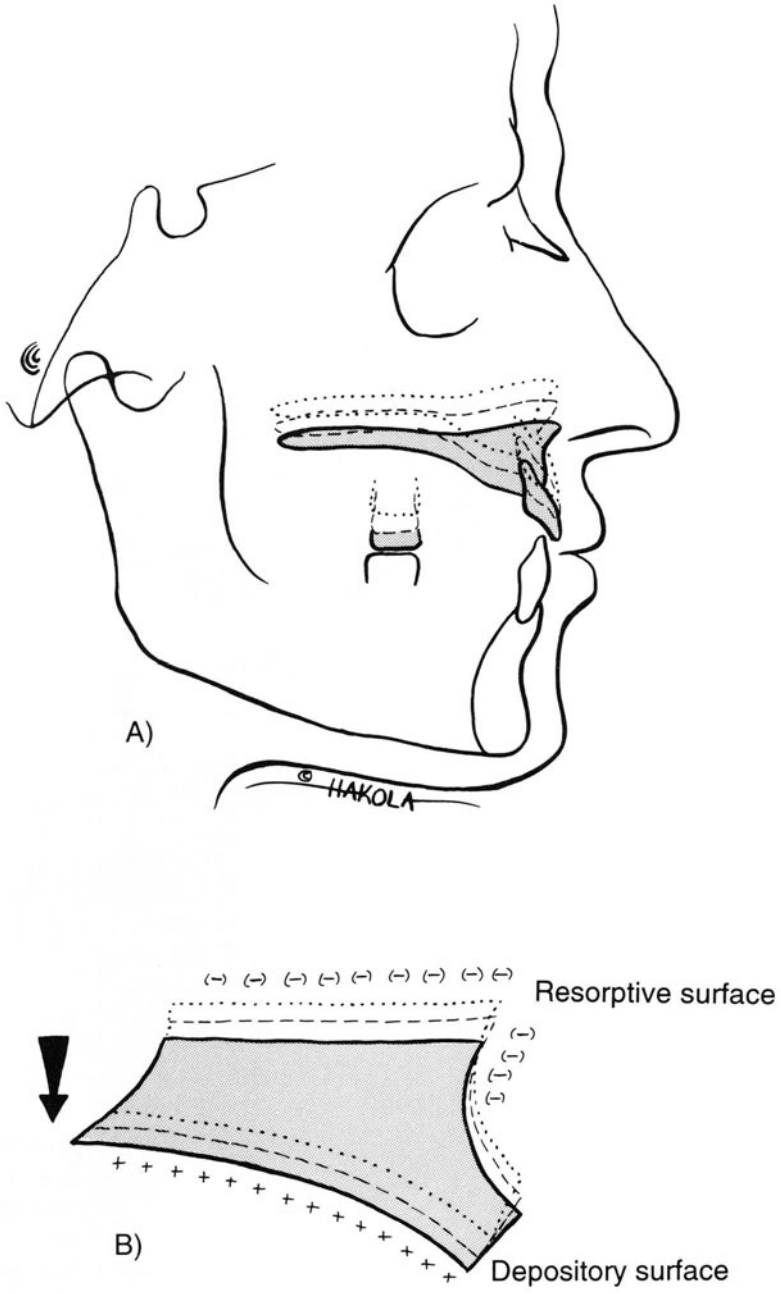

Figure 2.3. Relocation (progressive sequential movement) of the component parts as the palate (A, B) grows downward by periosteal resorption of the nasal side and periosteal deposition on the oral side.

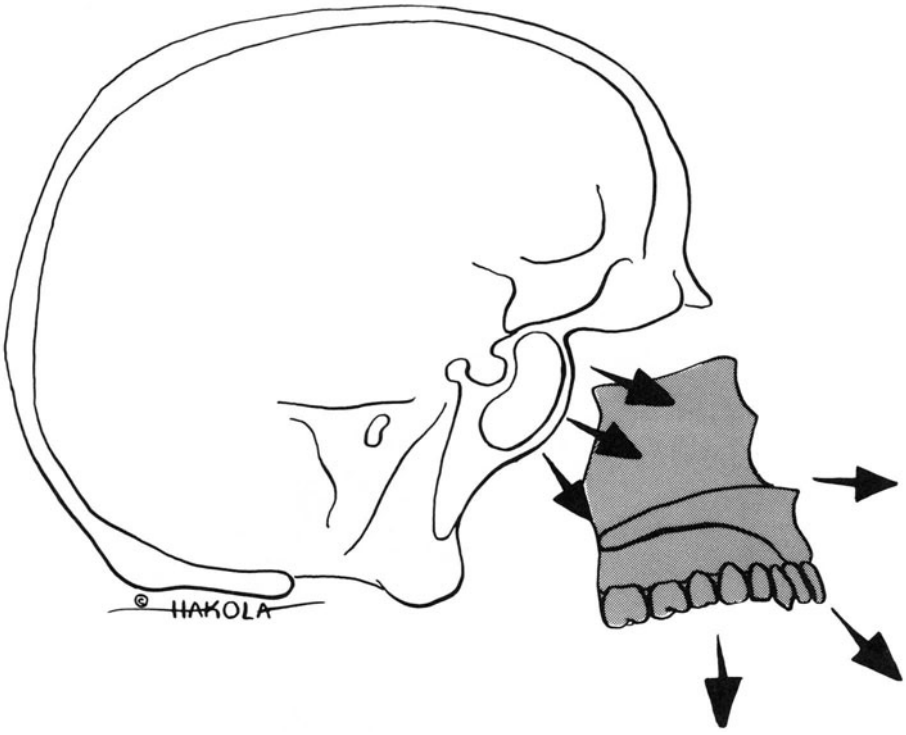

FIGURE 2.4. Secondary displacement: The movement of the whole bone caused by separate enlargement of other bones increases the size of the middle cranial fossa, resulting in a secondary displacement of the maxillary complex anteriorly and inferiorly.

action might augment these genetic determinants, morphologic features of bones (such as the mandible or maxilla) were thought to be largely self-determined. From these ideas arose the concept of *specific growth centers* that regulated the bone they subserved.[2] Today, most researchers discount the concept of growth centers, replacing it instead with the notion of *sites of growth* or localized regions within individual bones, operating under their own processes of control, with a feedback system that allows reciprocal adaptation with other sites.[2]

Nasal Septum Theory

Because sutures are traction-adapted types of tissue, they alone cannot drive the nasomaxillary complex downward and forward.[2] Scott proposed that because the cartilaginous nasal septum occupied a strategic position, it may be the "pacemaker" causing midfacial displacement as it grows in size.[4] Because cartilage is a more pressure-tolerant tissue than sutures, it was thought that it had the developmental capacity to push the nasal maxillary complex downward and forward.

Laboratory testing of Scott's *nasal septum theory* has been difficult to carry out. Latham speculated that the actual physical force for maxillary displacement may be the pulling action of the *septopremaxillary ligament* during septal enlargement, rather than a pushing action of the nasal septal cartilage itself.[5] Enlow reasoned that whether or not the nasal septum operates as an essential pacemaker for maxillary displacement, it is a component of the functional matrix and therefore contributes some share to craniofacial development.[2]

Functional Matrix Theory

Perhaps the most important conceptual advance in craniofacial growth was the realization that no single agent, such as the nasal septum, had sole responsibility for the entire growth process.[2] Moss advanced the *functional matrix theory*, which states that because bone and cartilage lack inherent growth potential and genetic control, growth is simply an adaptive response to the growth and function of the surrounding tissues.[6] In extirpating various normal cranial vault sutures in rats, Moss found that when one or more sutures was excised, growth of the calvarium was not altered.[2] Thus, expansive forces of growth did not seem to reside in the sutures themselves. Moss concluded that the calvarial bones lay within the encapsulating layers of soft tissue comprising a neurocranial capsule, which expanded as a whole in response to the growth of the enclosed brain, carrying the bone passively outward.[2] Secondary formation of bone occurred at the sutural edges.

Other Aspects of Growth Control

Growth control has been considered primarily a local process complimented by systemic support.[2] Growth is

carried out by regional fields of differing growth potential. The diverse cell populations within each of these fields responds to intracellular and extracellular signals. In the immediate environment enclosing an osteoblast or osteoclast, a first messenger hormone or enzyme, a bioelectric potential change, or a pressure/tension factor acting on the cell's outer membrane receptors can activate second messenger, membrane-bound adenyl cyclase.[2] Adenyl cyclase in turn accelerates the transformation of adenosine triphosphate (ATP) to cyclic AMP within the cytoplasm, which activates the synthesis of other enzymes relating specifically to bone deposition or resorption. Thus, the concept of *control messengers* and their importance in craniofacial growth control theories can be seen.[2]

Bioelectric signals also participate in the control of craniofacial bone growth.[2,7] Distortions of collagen crystals in bone caused by minute deformations due to mechanical strains presumably produced by muscular actions, generate bioelectric changes in the area of deformation.[2] These altered potentials appear to relate either directly or indirectly to the triggering of osteoblastic and osteoclastic responses. The complex interrelationships leading to growth and development of bones has been put in graphic form by Enlow (Fig. 2.5).

It is important to distinguish facial soft tissue development from that of bone. In studies of the facial soft tissue profile, certain assumptions have been made: (1) the soft tissue profile was closely related to its supporting bony structures; (2) changes in the skeletal profile bring predictable soft tissue changes; (3) bony profile landmarks are appropriate for determining soft tissue profile; and (4) changes in soft tissue profile can be studied using soft tissue landmarks alone.[8] Although these assumptions are correct to a certain extent, changes in body composition, such as in skin subcutaneous tissue thickness, also impact on changes in soft tissue profile. In all likelihood, a nonlinear relationship exists between craniofacial skeletal growth and associated changes in facial soft tissue profile.

Growth of Specific Craniofacial Regions

Growth of the craniofacial skeletal and soft tissues must be put into the perspective of overall growth and development of the body. Body proportions change considerably during fetal and postnatal life.[1] For example, during fetal life the head appears disproportionately large compared to the body. Beginning about eight months in utero, subcutaneous fat begins to accumulate and until birth, major changes in proportion are due to accumulation of fat.[1] The alteration of body configuration is a result of selected regional growth.[1] In infancy the head grows most rapidly, so that during the first year of life, head circumference is greater than chest circumference. After the first year, head growth slows down. Growth velocity is the rate of growth over a period of time. In general, growth is most rapid immediately after birth and then, between the ages of 2 and 12 years in boys, or 2 and 10 years in girls, growth velocity

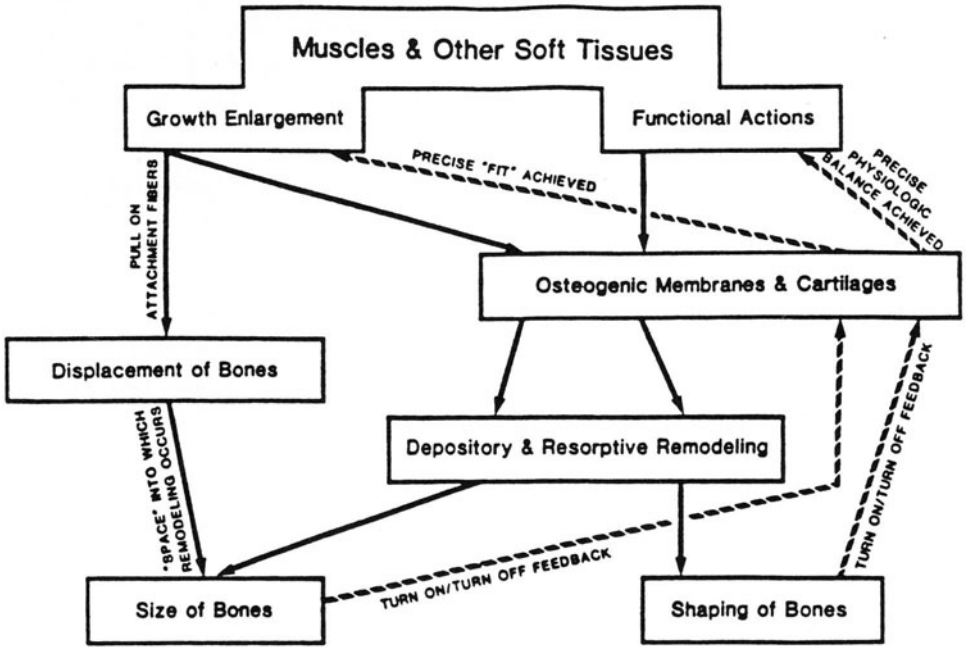

Figure 2.5. Graphic depiction of the complex interrelationships leading to growth and development of bones (Reprinted with permission from Enlow, D.H. Structural and functional "balance" during craniofacial growth. In L.W. Gaber (Ed.), *Orthodontics: State of the Art, Essence of the Science.* St. Louis: C.V. Mosby, 1986.)

slowly deaccelerates.[1] During adolescence, it increases again. The adolescent peak for girls is approximately 12 years and for boys approximately 14 years of age.[1] Figures 2.6 and 2.7 show the height velocity in males and females from ages 4 to 16. Weight velocity in general parallels height velocity.[1]

Calvarial Growth

The skull develops from mesenchyme surrounding the developing brain and consists of two parts—the neurocranium (the protection for the brain) and the viscerocranium (the main skeleton of the jaws).[1] The neurocranium is further divided into cartilaginous and membranous portions.[1] The cartilaginous neurocranium (or chondrocranium) consists initially of the cartilaginous base of the developing skull, which forms by fusion of several cartilates.[1] Later, enchondral ossification of the chondrocranium forms the bones of the face and the skull.[1] Intramembranous ossification occurs in the mesenchyme at the sides and top of the brain, forming the cranial vault or calvaria.[1] During fetal life the flat bones of the vault are separated by dense connective tissue membranes called sutures.[1] The six large fibrous areas where the sutures meet are called fontanelles (Fig. 2.8A).[1] The cartilaginous viserocranium consists of the cartilaginous skeleton of the first two pairs of brachial arches, which will ultimately form the middle ear ossicles, part of the hyoid bone, and the styloid process of the temporal bone.[1] Intramembranous ossification occurs within the maxillary and mandibular prominences (the first brachial arch) and subsequently forms the maxillary, zygomatic, and squamous temporal bones in the mandible.[1]

Postnatal growth of the skull occurs because the fibrous sutures of the newborn calvaria permit the skull to enlarge during infancy and childhood.[1] The increase in size is greatest during the first 2 years, the period of most rapid postnatal brain growth.[1] The calvaria normally increases in capacity until 15 or 16 years of age.[1] After this, any slight increase in size is due to thickening of the bones.

The overall head shape is closely related to the bony structure of the skull and to the shape of the underlying brain. It is well established that alternations in head shape can be the result of unusual brain growth, but they may also reflect a number of other factors such as premature synostosis of cranial sutures or unusual intrauterine mechanical forces. Abnormal planes of muscle pull, as in torticollis, may also cause asymmetric skull growth.

Five major sutures are present in the calvaria (Fig. 2.8B). Three (the coronal, lambdoid, and squamosal) are paired and two (the sagittal and metopic) are single. Cranial growth normally proceeds in a direction perpendicular to each of the major sutures. Increased length of the skull in comparison to width (dolichoecphaly) or the converse (brachycephaly) can be normal variants. However, both can also occur because of premature synostosis of cranial sutures (Fig. 2.9).

It is important to have a general understanding of the timing of normal fontanelle and sutural closure. With increasing age, the fontanelles and sutures become smaller and narrower due to the deposition of bone. There is considerable variation of the velocity of this process in different individuals and on the two sides of the same skull.[1] The anterior fontanelle usually is reduced to a fingertip size by the first half of the second year. The posterior fontanelle may close during the last 2 months before birth or the first couple of months following birth. The anterior lateral fontanelle disappear during the first 3 months and the posterior lateral fontanelle during the second year of life. The metopic suture begins to close in the second year and is usually obliterated during the third year of life. It persists throughout life in about 10% of individuals. The great sutures of the vault (coronal, lambdoid, sagittal) persist normally throughout infancy and childhood and do not completely close before the third decade.

Head circumference and brain weight peaks at approximately 1 to 2 years of age and then gradually begins to taper off.[1] As the rapid expansion of the brain slows, continued growth by remodeling with endocranial resorption and ectocranial deposition of new bone continues to provide for the slower and more gradual development of the calvarium.[1] Thus, the osseous drift produced by such remodeling produces physical migration of the skull table.[2] This process of remodeling involving periosteal resorptive surfaces, first begins in the fetus at approximately 10 weeks of age in two principal locations: (1) the lining surfaces of the bone around tooth buds, and (2) the endocranial surface of the frontal bone.[2]

Remodeling, therefore, is a process that actually accompanies growth, from the start, becoming the principle mode of bony growth in the calvarium as sutural fusion takes place.[2] In the postnatal skull, the entire dural surface from the frontal bone to the supraorbital rim is resorptive.[2] On the orbital side beneath the orbital rim, the surface is depository medially, and resorptive laterally.[2] The roof of the orbit is entirely resorptive on the dural side and depository on the orbital side.[2] Differences between prenatal and postnatal patterns of growth and remodeling in the thinly layered roof of the orbit and floor of the anterior cranial fossa appear to be based on differential growth rates by the eyeballs and the cerebral hemispheres.[2] The relative rate of eyeball growth is fastest before 24 weeks gestation.[2] As the eyeballs pass their stage of maximum expansion and the frontal lobes begin theirs, a reversal in the growth pattern occurs.[2] Development of the bony orbit is therefore dependent on both the growth of the frontal lobes and

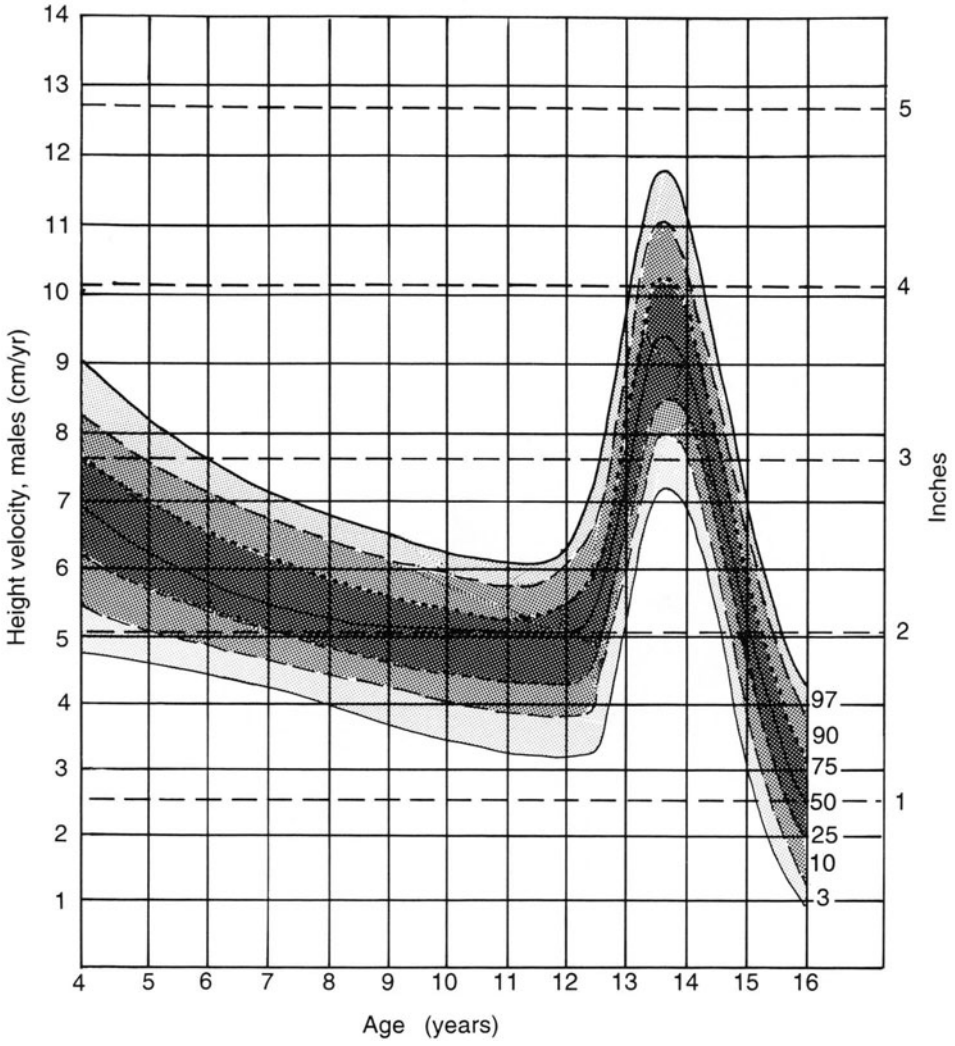

Figure 2.6. Height velocity, males.

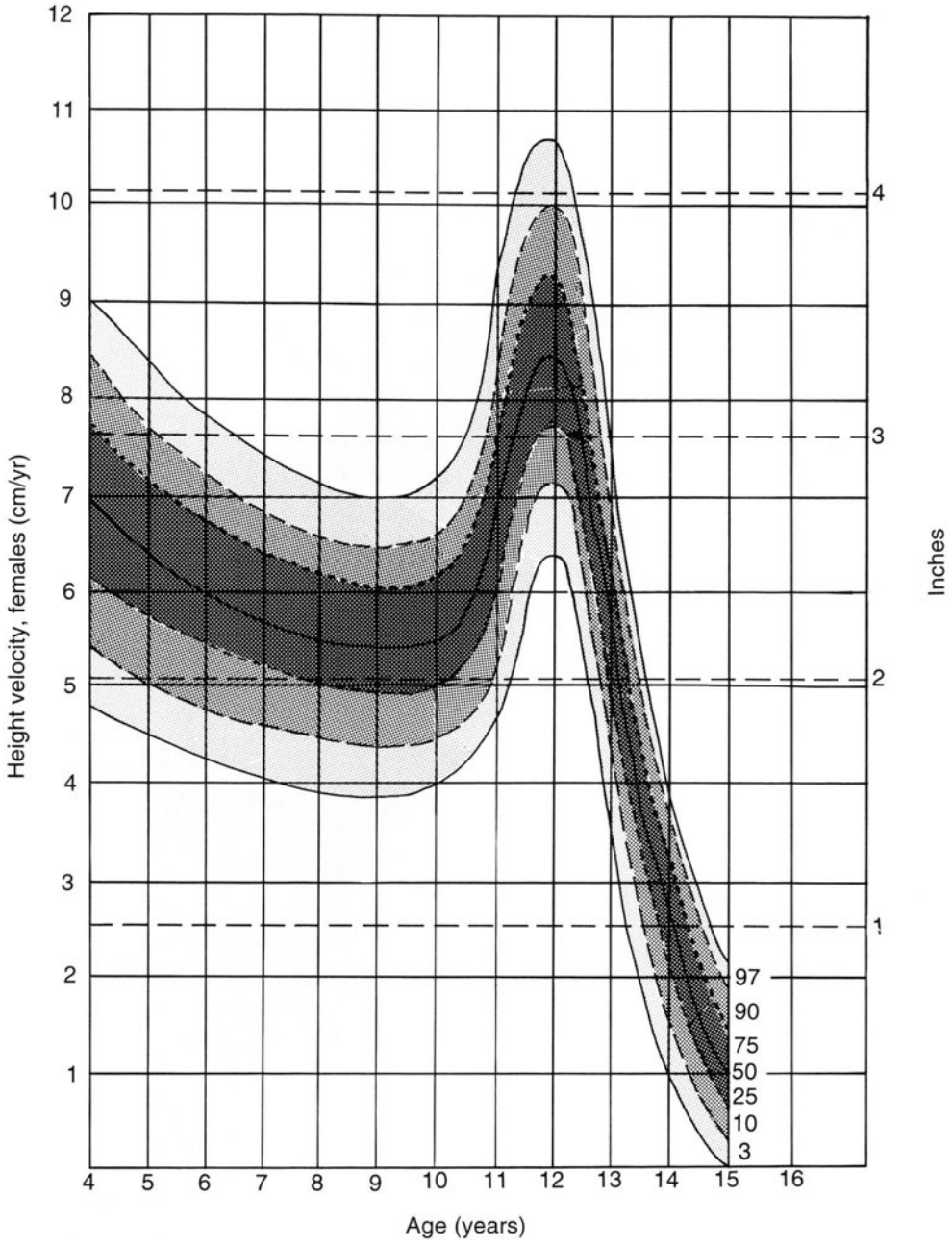

Figure 2.7. Height velocity, females.

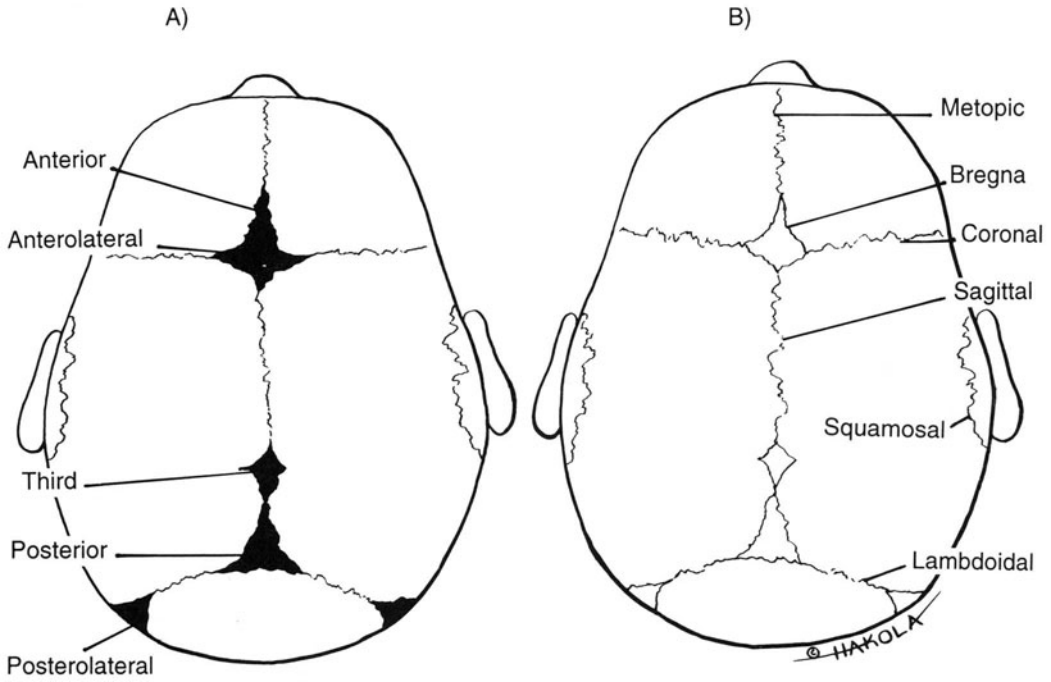

Figure 2.8. Normal fontanelle and suture landmarks. (A) Fontanelles. (B) Sutures.

growth of the orbital contents. The bulk of both orbital and cerebral growth slow considerably by 6 years of age.[2]

Growth and development of the forehead and supraorbital ridge in the adult occurs by expansion of the middle cranial fossa with subsequent anterior displacement of the anterior cranial fossa.[2] This process is initially associated with early rapid growth of the brain and hence, the high protruding forehead of the infant. In the infant, the flat supraorbital ridge blends into the forehead, lying on a vertical plane with the inferior orbital rim. With subsequent growth of the nasomaxillary structures, the forehead becomes less prominent, but the supraorbital ridge tends to protrude. The medial lateral walls of orbit diverge with growth, with the medial wall extending anteriorly as the nasomaxillary complex grows, while the lateral wall of the orbit and zygoma remodel posteriorly. These changes produce the characteristic superior medial protuberance of human orbit.

Development of the *paranasal sinuses* not only influences the development of the nose and maxilla, but also the forehead and glabella region. The ethmoid or maxillary sinuses begin to evaginate from the nasal cavity in the second trimester.[8,9] They first present as separate recesses, with the ethmoid sinuses later growing into a honeycomb of cells. The growth of the maxillary sinus parallels that of the face. Maxillary sinuses are narrow in the newborn and do not reach the area beneath the orbit. The sinus slowly increases in size and becomes quite large after 5 years of age. During the third and fourth years, the medial and lateral dimensions of the maxillary sinus increase considerably. By age 5, they extend to a point lateral to the infraorbital canal. It is only after the eruption of permanent dentition in the 12th year and the development of the alveolar process that the maxillary sinus descends below the level of the nasal floor. Eruption of the permanent maxillary teeth determines the inferior growth of the maxillary sinus and therefore, the level of the sinus floor. By the age of 8 years, the floor is at the level of the inferior maxillary sinus, and is fully developed by age 16.[8,9] The frontal sinus also develops as an evagination from the nose during the second trimester.[9] It can be distinguished from the ethmoid sinus after 5 years of age.[9] The sinus reaches adult size by late puberty. The frontal sinuses are seldom symetric and vary in size and shape. Occasionally, they may be absent.

What happens in the cranial base also effects the ultimate structure of the face. The cranium may be considered the template upon which the face develops.[2] As each temporal lobe of the cerebrum grows, the middle cranial fossa correspondingly expands. The bony surface of the whole cranial floor is predominantly resorptive. This is in contrast to the endocranial surface of the skull roof, which is predominately depository.

The midline of the cranial base is characterized by the presence of synchondroses. These are remnants from the primary cartilage of the early cartilaginous base of the cranium after the enchondral ossification centers appear during fetal development.[2] A number of synchondroses are operative during the fetal and postnatal

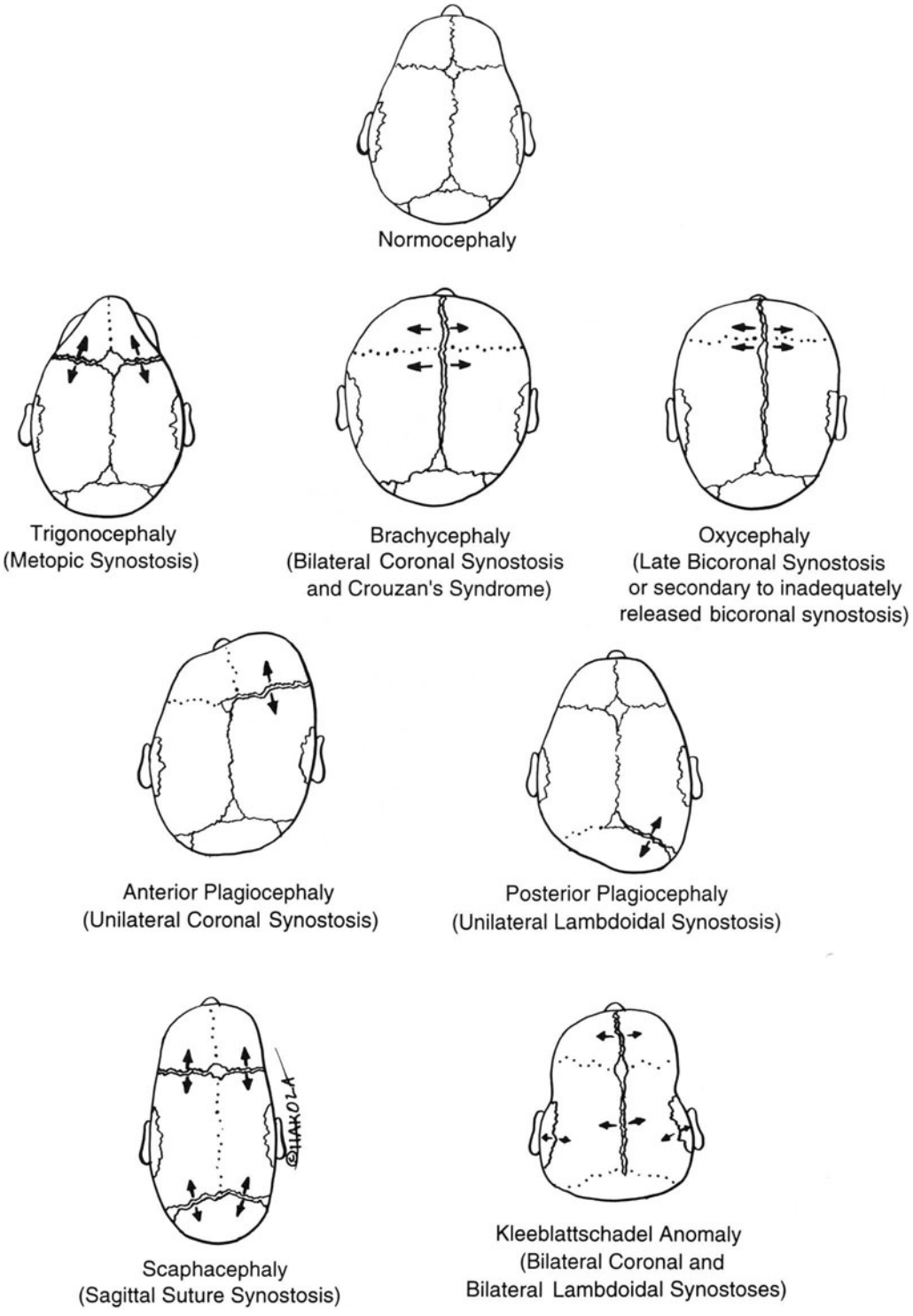

Figure 2.9. Abnormal patterns of sutural fusion.

periods. During childhood the *spheno-occipital synchondrosis* becomes the principal growth cartilage of the cranial base (Fig. 2.10A).[2] This spheno-occipital synchondrosis provides a pressure-adapted growth mechanism, which contrasts with the tension-adapted sutural growth process. Compression is involved in the cranial base, which unlike the calvaria, supports the weight of the brain and face.[2] This spheno-occipital synchondrosis is retained throughout childhood and ceases to be active at 12 to 15 years of age. The spheno-occipital segments then become fused in the midline area before about 20 years of age.[2]

The presence of the spheno-occipital synchondrosis provides for elongation of the midline portion of the cranial base by a pressure-adapted mechanism, and its gradual enchondral ossification (Fig. 2.10B).[2] Enchondral bone growth by the spheno-occipital synchondrosis produces primary displacement of the bones involved. Thus, the sphenoid and occipital bones move apart. At the same time, new bone is laid down. The interior of the sphenoid bone gradually becomes hollow to form the sizeable sphenoid sinus. This sinus lies just behind, and in a direct line with the bony nasal septum of the nasomaxillary complex. As the midface is displaced forward and downward, the sphenoidal sinus is drawn outward by the enlargement of the body of the sphenoid. With the lengthening and flexure of the cranial base, and the enlargement of the anteroir cranial fossa by expansion of the frontal lobes, the maxilla is moved forward and displaced downward.

Expansion of the middle cranial fossa has a major secondary displacement affect on the anterior cranial floor, nasomaxillary complex, and the mandible.[2] As horizontal enlargement of the middle cranial fossa and brain growth advance the nasomaxillary complex by forward displacement, the horizontal span of the pharynx correspondingly increases. The skeletal dimension of the pharynx is established by the size of the middle cranial

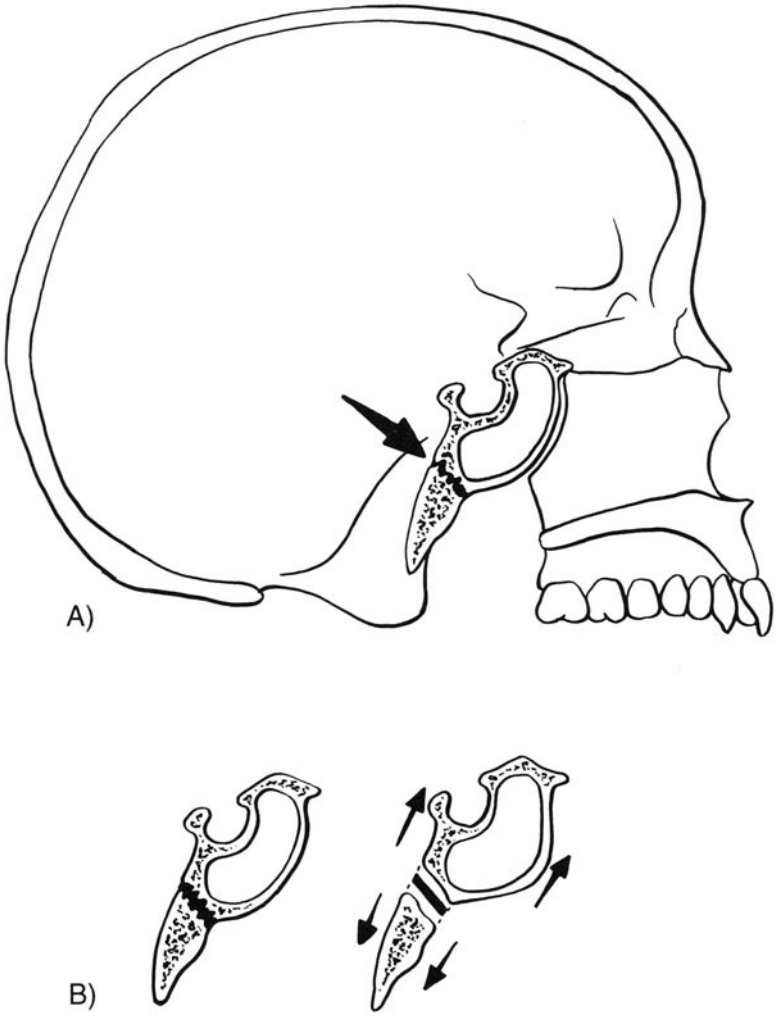

Figure 2.10. (A) The principal growth cartilage of the cranial base is felt to be the spheno-occipital synchondrosis, whose location is depicted by the arrow. (B) Direction of growth of the shpeno-occipital synchondrosis.

fossa. The ramus of the mandible bridges the pharynx and as this space enlarges, the ramus increases an equivalent amount. With continued growth, the whole mandible is displaced downward and forward.[2]

The facial bones continue to grow until approximately 21 years of age in the male. Much of the growth, however, is completed well before the teen years. The orbits, for example, have usually obtained their full adult size by 7 years of age and are similar in dimension to the adult by the age 2 years. In contrast, the nasal bone demonstrates major growth in adolescence.

The major proportion of cranial growth is completed by age 2.[1] From birth to age 10, the brain triples in size and is 90% of its final mass by the age of 10. The peak rate of growth in the head and face occur between 3 and 5 years of age.[1] After this, growth proceeds slowly, but an acceleration occurs again between the ages of 13 and 15.[1] Growth is greatly diminished after the age of 15.

Growth of Basicranial, Nasomaxillary, and Mandibular Complexes

As eluded to throughout this chapter, the major components of the cranial base, ethmoid, and maxillary region, and the mandible, are not independently assembled. Close structural innerrelationships exist for all the bones of the face. The relationship between the cranial base and ethmomaxillary complexes are closely bound because of the direct union by shared sutures between them.[10] The mandible is less constrained by the boundaries of growth fields established by the basicranium because it is a separate bone not sharing direct articulation with many common sutures.[10] Shown in Fig. 2.11 are the counterpart relationships among the various parts of the face and cranium. Note also that the ethmoid-maxillary complex relates to the anterior cranial fossa and the body of the mandible relates to the corpus of the maxilla, and therefore in turn to the anterior cranial fossa.

As expansion of the frontal lobes of the cerebrum is simultaneously accompanied by a corresponding enlargement of the anterior cranial fossa, the entire ethmoid-maxillary complex is displaced anteriorly (Fig. 2.12A). Growth simultaneously proceeds posteriorly to keep the posterior aspect of the maxilla in line with its prescribed anatomic boundary. The extent of backward growth approximately equals the amount of forward displacement (Fig. 2.12B).[10] As mentioned earlier in the text, the source of biomechanical force responsible for

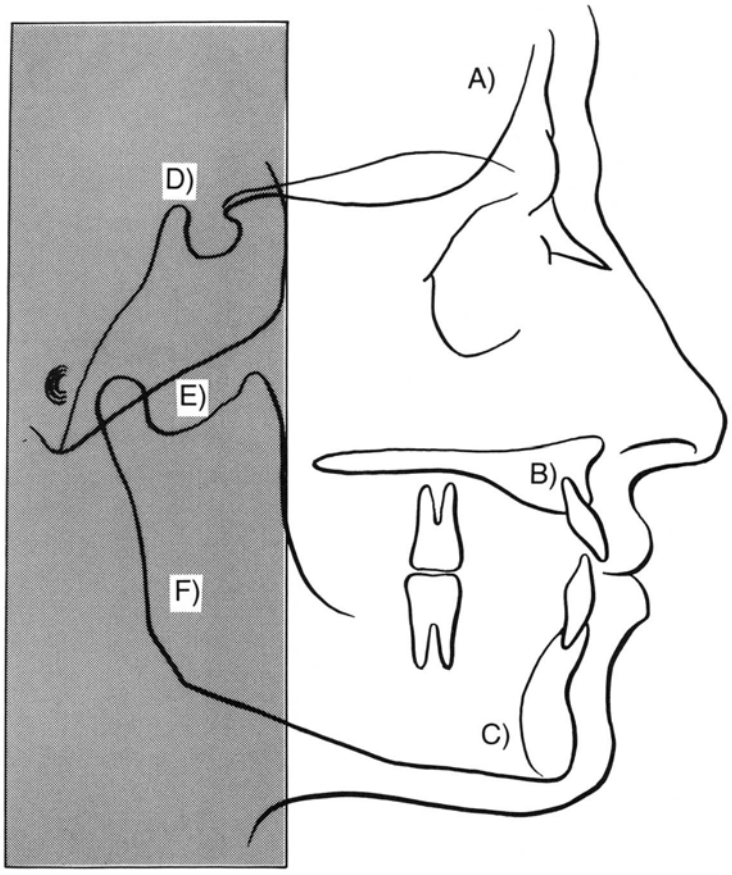

Figure 2.11. Relationship among various parts of the face and cranium. The anterior cranial fossa (A); the palate and maxillary arch (B); and the mandibular corpus (C); function as structural counterparts to one another. The middle cranial fossa (D); the postmaxillary (pharyngeal) compartment (E); and the mandibular ramus (F); also have counterpart relationships.

the displacement of the maxilla is a controversial issue. Currently, it is thought that maxillary displacement results from a composite expansion of all facial soft tissues (the functional matrix), which serves to carry the entire midface downward and forward.[6]

As the maxilla lengthens horizontally by backward growth at the maxillary tuberosity, the mandibular arch lengthens posteriorly at this counterpart structure, the lingual tuberosity. Posterior lengthening of the bony mandibular arch is thought to proceed by continuous remodeling conversions from the region of the ramus.[10] Thus, the ramus moves backward by combinations of deposition on its various posterior-facing surfaces and resorption from anterior-facing surfaces. As the entire

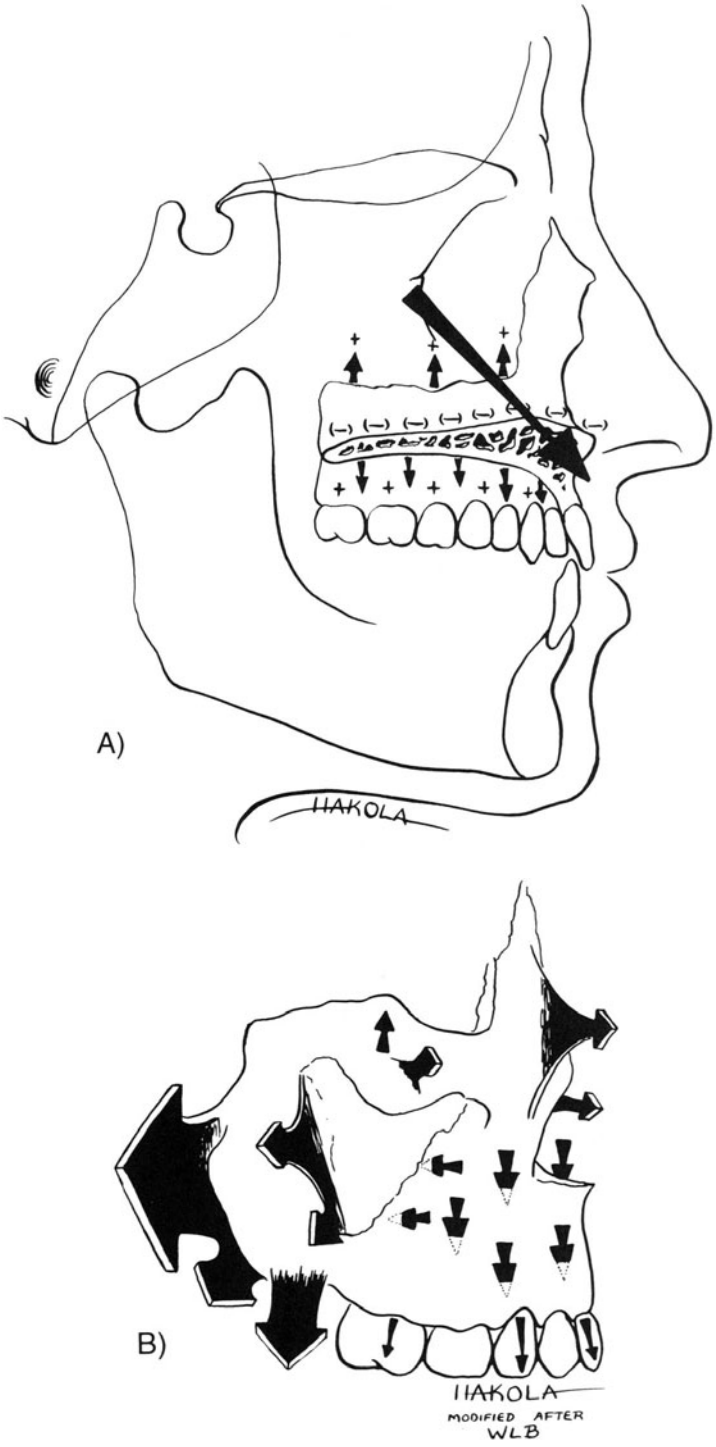

Figure 2.12. Palatal and orbital growth. (A) Downward displacement of the maxilla associated with an upward growth of the orbital floors. This maintains the orbits above the level of the nasal cavity. (B) As the maxillar grows down and forward by maxillary sutural growth, there is appositional and resorptive change in the palate and orbital floors which give this area its final position.

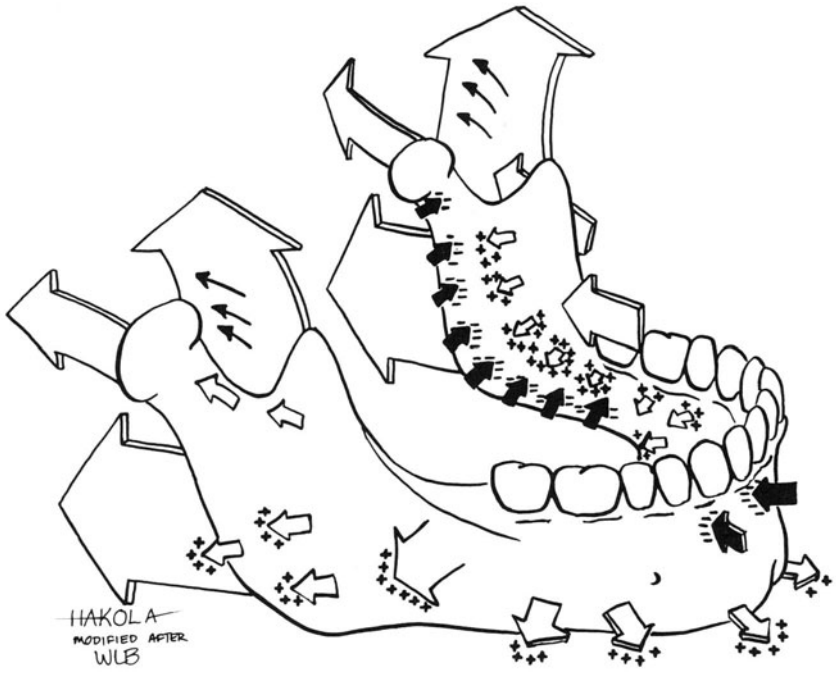

Figure 2.13. Multidirectional mandibular growth.

ramus relocates posteriorly, areas once occupied by the ramus become remodeled into additions, therefore lengthening the body of the mandible (Fig. 2.13).

The condylar component of ramus growth has historically received special emphasis because it was once felt to represent the pacemaker of mandibular growth.[10] The condyle is now believed to be the source of mechanical thrust producing anterior inferior displacement of the mandible.[10] Another role the condylar cartilage provides relates to the construction and growth potential of the cartilage itself.[10] Chondrogenic proliferation takes place in a manner that differs from most of the other growth cartilages in the skeleton. The continued growth of the condyle is not committed to a one-direction course. It can significantly alter the route of its growth to provide the best compromise for its multifaceted junction with the cranial base.

Remodeling takes place throughout all parts of each of the bones making up the midfacial complex.[10] Remodeling growth in the nasal maxillary complex involves relocation of its various parts into new positions by progressive desposition and resorption.[10] The lining surface of each nasal chamber is largely resorptive in nature, therefore expanding the size of the airway.[10] As the nasal side of the hard palate undergoes resorption, the oral side undergoes progressive deposition of new bone.[10] The maxillary sinuses expand in all directions except medially by a simular combination of internal bone removal and external addition.[10]

Abnormalities of Craniofacial Growth—Clinical Correlations

Cleft Lip and Palate

The oral facial cleft not only interferes with development and morphology of the maxilla but also causes alterations in contiguous structures that are developmentally normal.[11] Ross appropriately notes that it would be helpful in treatment planning if one could distinguish between structures that are intrinsically abnormal and those that only appear to be abnormal because they have become distorted by secondary environmental disturbances.[11] The former are resistant to correction, whereas the latter may be self-correcting if the proper environment is provided. Ross feels that the embryo with a cleft of the lip and palate has basically an intrinsically normal facial framework to which is attached a midly deficient maxillary complex.[11]

The general growth stature of children in cleft lip and palate is smaller than that of children without clefts. Shibasaki and Ross found evidence that the pubertal growth spurt of facial structures is delayed in patients with isolated cleft palate.[12] Studies by Graber,[13] Ortiz Monasterio and associates,[14] and others indicate that the basal maxilla achieves normal relationships with the remainder of the face in the unoperated adult with unilateral cleft lip and palate. This suggests that growth is not ultimately inhibited by the presence of a cleft.

Patients with repaired cleft lip and palate do however, show facial growth deficits that can be associated with severe functional and aesthetic problems. It is thought that surgery is mainly responsible for these adverse facial and dental sequela.[11]

Cephalometric studies consistently show that there is some change in the position of the mandible.[11] The chin is usually retruded and there is greater facial height in spite of normal or decreased maxillary height. The mandible is more open than in the normal individual, and thus, the chin point moves downward and backward while the mandibular plane becomes steeper.

Maxillary growth in children with cleft lip is minimally disturbed.[11] There may be dental irregularities in the region of the clef and positional differences in the anterior nasal spine.[11,15] Facial form in individuals with isolated clefts of the hard and soft palate is characterized by a deficiency in maxillary length and retrusion of the anterior maxilla relative to the cranial base.[11] These characteristics become accentuated as the patient matures. The causes of abnormal facial growth in patients with cleft lip and palate are multifactorial.[11] The effect of lip reconstruction is generally thought to be beneficial. The soft tissues and underlying muscles are reapproximated, producing a normal functional matrix to influence maxillary growth. However, when the lip is globally deficient and repaired under tension, increased pressure on the basal maxilla may lead to excessive molding of the dental alveolar structures.

The affect of early repair of the cleft alveolus with bone grafts is also controversial in terms of facial growth. Friede and Johanson reported that patients who had received their alveolar bone graft in infancy developed severe maxillary retrognathia and vertical deficiency that worsened with age.[16] Rosenstein and associates, however, continue to use primary bone grafts and after long-term follow-up, only 22% of their patients with unilateral cleft lip and palate required orthognathic surgery.[17] Ross pointed out that the alveolus and portions of the maxilla in which the bone graft is placed is not a primary site of maxillary growth.[11] Consequently, he rationalized that anterior posterior maxillary development should not be affected if grafts are confined to the alveolar area, especially when performed during the stage of mixed dentition.

In obtaining mucoperiosteal tissue to close the cleft, areas of denuded bone adjacent to the alveolar process may be left. Scar tissue, which fills in these regions, exerts an initial contracting force on adjacent tissues. Proponents of less traumatic surgery suggested that the soft palate be repaired to facilitate speech, but surgical correction of the hard palate be delayed for later repair to prevent maxiallary growth abnormalities. Although results have been controversial, speech pathologists have been less than enthusiastic about the results of delaying surgery past the age of early speech development.[18]

Hemifacial Microsomia

Hemifacial microsomia (HFM) is a variable, progressive and asymmetric craniofacial anomaly. HFM involves the skeletal, soft tissue, and neuromuscular components of the first and second branchial arches and is the second most common congenital facial anomaly after cleft lip and palate. In the mildest form, the change in the condylar cartilages is minimal although the disc may be missing.[19] This may be determined radiologically by the clear cortical outline of the condyle and reduced joint space. The shape of the condylar head is normal, but small. The glenoid fossa may be shallow or missing. The joint function is generally adequate and asymptomatic. The side of the mandible grows, but less so than the normal, presumably because condylar cartilaginous proliferation does not occur.[19]

The next degree of mandibular involvement is associated with a hypoplastic temporomandibular joint with cone-shaped condylar process and an attached lateral pterygoid muscle. When the condylar process is missing and the lateral pterygoid muscle is either absent not attached, and there is no glenoid fossa, slope, or eminence, the body of the mandible with its alveolar process and developing dentition will not disocclude from the maxilla.[19] Consequently, the down and forward movement of the maxilla becomes inhibited. The result is canting of the occlusal plane and impaired mesial migration of the maxillary teeth on the affected side. In the most severe case, the mandibular involvement consists of absence of both the condylar and coronoid processes, resulting in absence of the entire ramus. In this condition, only rudiments of the masseter, medial and lateral pterygoid, and temporalis muscles are present. The bony development of the body of the mandible is limited to alveolar bone surrounding tooth buds as they form.[19]

Several treatment regimens have been employed in the growing child and take advantage of increasing the size of the mandible in an effort to prevent secondary deformities of the maxilla. In addition, the underdeveloped soft tissues are treated, and articulation between the mandible and temporal bone is created if such an articulation is missing and a functional occlusion with good aesthetic appearance is achieved. It has been demonstrated by some authors that if mandibular lengthening and creation of an open bite are done early in the mixed dentition stage, the vertical growth potential of the midface can be used to minimize secondary deformities of the maxilla and often prevent the need for a later maxillary osteotomy.[19] The Boston Children's Hospital group and the San Francisco group have demonstrated vertical growth in the midface, which closed the surgically created open bite and leveled the occlusal plane without a maxillary osteotomy in selected cases.[20] Similarly, the San Francisco group reported continued growth in the length of the mandibular ramus in the

majority of their patients operated on early in their growth period.[21]

Craniosynostosis

Skull morphology and cranial base changes have been studied following treatment of craniosynostosis. Marsh and Vannier used three-dimensional osseous surface images from CT scans to evaluate the endo- and ecocranial bases of 87 patients with a variety of craniosynostoses.[22] They found that cranioorbital surgery in infancy for nonsyndromal solitary and bicoronal synostosis seemed to induce normalization of endocranial growth in the first postoperative year. This normalization occurred to a lesser degree in patients with multiple synostoses. It was unclear whether persistent postoperative dysmorphology of the cranial base reflected abnormal rather than osseous growth. Friede et al. also evaluated skull morphology after early craniotomy in patients with premature synostoses of coronal suture.[23] Cranial height in particular was increased, especially in patients with Crouzon's or Apert's syndrome following operation.

One should realize, however, that little information is available on regional changes in growth following craniofacial surgery, which appear to be disturbed to some extent in almost all patients after surgery.

Maxillary Growth Following Total Septal Resection and Correction of Orbital Hypertelorbitism

Lejoyeux, Tulasne, and Tessier surveyed eight cases of orbital hypertelorbitism in whom total resection of the nasal septum had been performed.[24] It was interesting to note that these patients followed, rather closely, "normal" facial growth patterns with good stability of the facial axis and remarkable likeness to the predicted form of the mandible. The main difference observed between the postoperative control radiographs and growth forecast tracings in cases of septal resection was located in the premaxillary area, which seemed to exhibit reduced forward growth. This is in contrast to the accepted standard of nonmanipulation of the septum until after the adolescent growth spurt of the nose.

Facial Development

Branchial Arch and Branchial Cleft Derivates

While the development of the cranial vault, brain, cranial nerves, spinal cord, and cervical nerves is in progress, the cervicofacial structures and organs of special senses are also in the process of developing.

The fourth gestational week is characterized by the development of four paired branchial arches. (Actually five arches are formed, but the fourth and fifth arches fuse into a common arch.)

The arches consist of an ectoderm, mesoderm, and endoderm (see Fig. 2.14). The arches are delineated from each other by invaginating ectodermal branchial grooves, which are complimented by evaginating endodermal pouches produced in the primative pharynx. These pouches expand outward toward the branchial grooves. The soft tissue and skeletal structures of the head and neck develop from these branchial pouches, grooves, and arches.

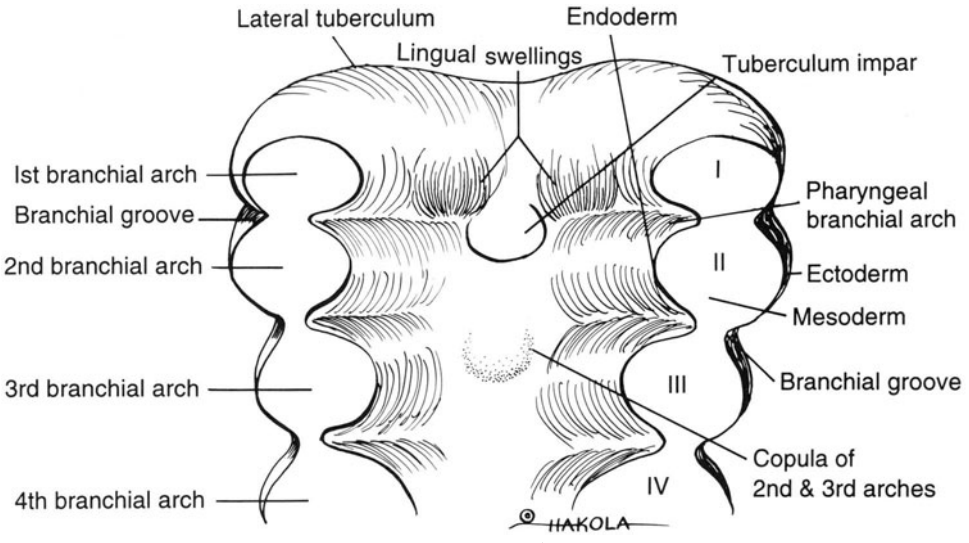

FIGURE 2.14. Branchial arches and pharyngeal arches at about 4 weeks in utero.

The majority of facial development takes place between the fifth through eighth gestational weeks (Figure 2.15). The facial structures are primarily formed by the paired first branchial arches and the so-called frontal nasal process, which appear to be the driving force or growth organizer in the development of the upper and midface. The frontal nasal process consists of the primitive forebrain and central midfacial elements. The optic cups (eyes) and olfactory pits (nose) develop in close association with the first branchial arch and frontal nasal process.

At five gestational weeks the first pair of bronchial arches are seen below and in front of the developing forebrain.

As the opposing branchial arches grow ventrally forward toward the new facial midline, the first branchial arch splits into a mandibular and a maxillary process. The frontonasal process grows ventrally caudal until it meets and fuses with the maxillary process of the first branchial arch. The largest portion of the frontonasal process becomes the forehead. The triangular segment of the process becomes the root dorsum and tip of the nose, while the medial and lateral walls of the nasal pit become the medial and lateral nasal process, respectively. The lateral nasal process develops into the lateral nasal segments, the ala, and the nasal and lacrimal bones. The median processes are responsible for the collumella, membranous septum, and the prolabial and premaxillary segments. The primary palate is therefore a derivative of the median nasal process segments of the frontonasal process. Initially the forebrain is located ventrally forward in the frontal nasal process but retracts through the so-called foramen cecum at the root of the nose as the cranial vault forms around it. Failure of the forebrain to fully retract may result in a frontal encephalocele containing brain meninges and glial tissue bulging forward at the root of the nose, often displacing the orbits laterally into a hyperteloric configuration. Partial retraction of the forebrain may also lead to a menegeocele or a glioma, palpable as a mass anywhere along the midline nasal dorsum. The menengeocele, although it contains no brain tissue, may still connect to intracranial structures through a cranial defect, while a glioma represents an isolated or pinched-off segment of neural tissue without intracranial connection. Accordingly a CT scan is strongly recommended for the evaluation of midline frontal nasal masses.

The failure of proper frontonasal development may be represented by a central oral cleft notable for its lack of premaxillary or prolabial segments. This condi-

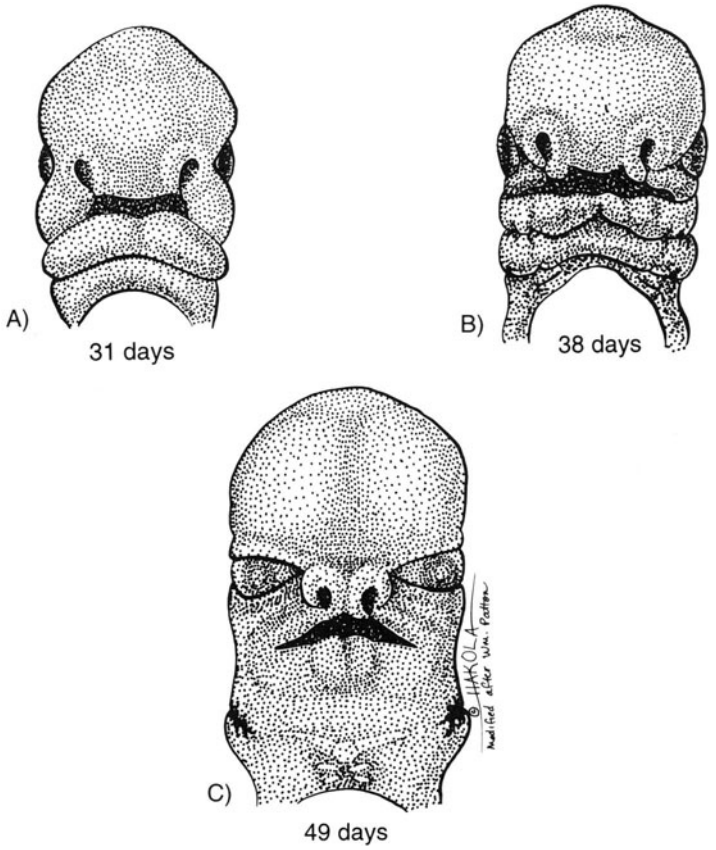

Figure 2.15. Embryological facial development (A, B, C).

tion, known as holoprosencephally, is associated with hypotelorism and serious forebrain deficiencies that limit the life of the infant.

As the frontonasal process grows forward, the paired maxillary and mandibular processes of the first branchial arch proceed to grow ventrally toward the developing facial midline. The mandibular processes meet and fuse to each other first. Shortly thereafter the paired maxillary processes fuse with the median nasal process. Mesodermal ingrowth is required for proper fusion between the processes. The failure of such ingrowth is the origin for facial and lip clefting, which is manifested along the embryologic fusion planes.

The Mandible

The mesenchymal core of the mandibular process of the first branchial arch is transformed into a cartilaginous bar called Meckel's cartilage. This cartilagenous structure does not form any part of the adult mandible but does play a roll as a template for the formation of the lower jaw. Meckel's cartilage extends dorsally into the tympanic cavity of the ear where it becomes surrounded by the temporal bone. The dorsal end of each cartilage is replaced by two auditory ossicles; these are the incus and the malleus. Ventrally the cartilage and its sheath convert into the spheno-mandibular ligaments. The main mass of this cartilage is replaced by membranous bone, which develops ventrally in the body of the future lower jaw and encloses both Meckel's cartilage and the inferior alveolar nerve. Dorsally in the ramus of the developing mandible the membranous bone takes the form of a plate that lies lateral to the cartilage and nerve. This relation explains both the position of the adult mandibular foramen and where the inferior alveolar nerve enters the foramen. Boney fusion of the two halves of the mandible does not take place until well after birth. The newborn infant has connective tissue and cartilage on either side of the midline symphysis, which only later is replaced by bone.

Development of the Tongue

The tongue is derived from the first, second, and third branchial arches. Accordingly, the cranial nerves of each of these arches is present in the developed tongue. The first branchial arch is represented by the mandibular division of the facial nerve (CN V^3), which provides general sensation to the anterior two thirds of the tongue. The second branchial arch is represented by the chorda tympani from the facial nerve (CN VII) providing taste to the anterior two thirds of the tongue. The third branchial arch is represented by the glossopharyngeal nerve (CN IX), which supplies taste and general sensation to the posterior one third of the tongue.

Between the fourth and fifth gestational week a median elevation appears in the floor of the primitive pharynx. This swelling, derived from the second branchial arch, is the median tongue bud or tuberculum impar. Rostral (what will become anterior in the mouth) to this median swelling develops two lateral swellings from the first branchial arch. These lateral lingual swellings overgrow the median tongue bud and fuse with each other forming the anterior two thirds of the tongue. The fusion line of these lateral swellings is represented by the median sulcus of the tongue while the median tongue bud virtually disappears.

The posterior one third of the tongue begins as the fusion of two second branchial arch derivatives called the cupola, which is caudad to the foramen cecum. The cupola is overgrown by the hypobranchial eminence, a third branchial arch derivative, which develops caudad to the cupola.

The tongue musculature is derived from myoblasts from the occipital somites, innervated by the hypoglossal nerve (CN XII). (See Fig. 2.16.)

The Palate

As the maxillary process grows forward to fuse with the frontal nasal process the olfactory pits develop into blind sacs, which overlie the roof of the front part of the primitive mouth. These primitive choanae become separated from the mouth by the subsequent development of the palate. The choanal sacs continue to develop posteriorly, to open as the secondary choanae into the pharynx. Failure of this opening to take place is referred to as choanal atresia, characterized by an incomplete nasal passage at birth.

The formation of the secondary palate takes place between the 8th and 12th gestational weeks. Initially, the paired palatine processes of the maxilla are vertically oriented along each side of the primitive oral cavity. Between the palatine processes lies the newly formed tongue. To properly form the palate these vertically oriented plates must migrate to a horizontal position and fuse at the midline. For this to take place, the tongue must clear from between the two palatine processes. As the embryo develops, the neck slightly extends allowing for the forward growth of the mandible. The mandible's forward growth clears the tongue from between the palatal processes and allows for their horizontal and midline migrations.

The failure of mandibular growth, as in the Pierre–Robin sequence, or the inability of the neck to extend, as in Klippel–Fiel syndrome, results in the inability of the tongue to clear the palatal processes and predisposes to clefts of the palate.

Fusion of the secondary palate at midline takes place from anterior to posterior (i.e., from the incisive foramen to the uvula). Bony formation and fusion only takes place in those areas that are in contact with the nasal septum. Where no septum is present there is no bone

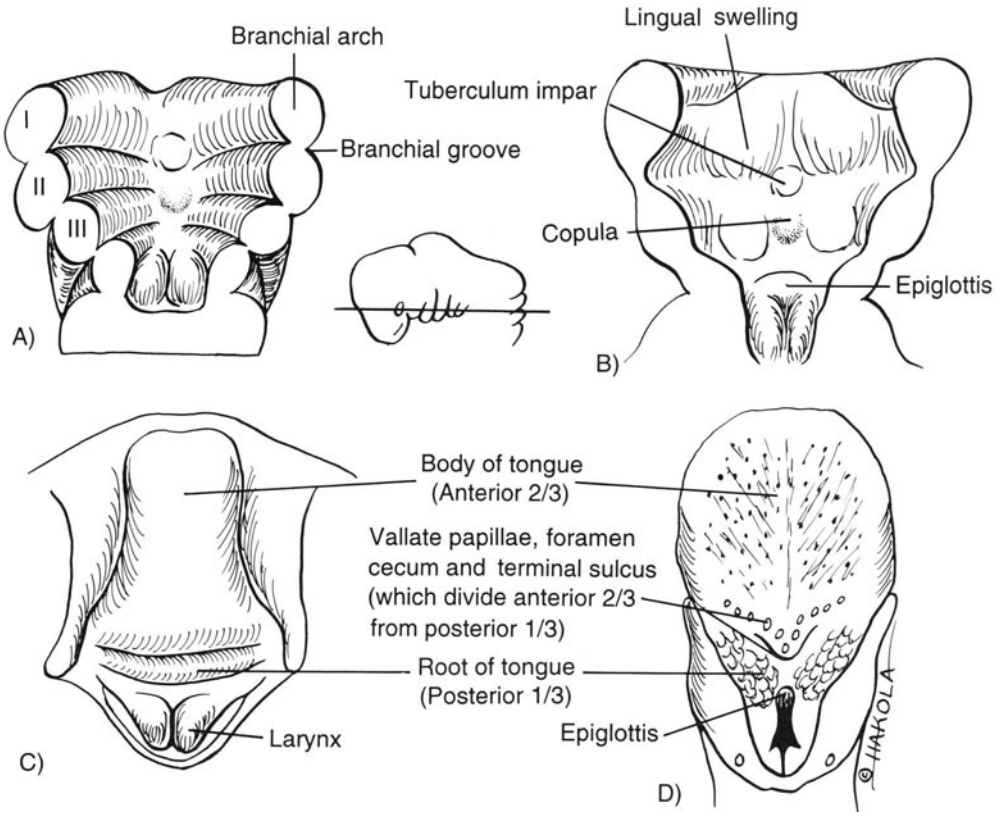

Figure 2.16. Development of the tongue (A, B, C, D).

formed, and instead one has the formation of the soft palate and uvula.

One must remember that the above description of palatal formation refers only to the secondary palate. The primary palate (i.e., that portion of the hard palate anterior to the incisive foramen), is formed from the medial nasal processes of the frontal nasal process. This primary palatal segment includes the premaxilla; alveolar processes on each side complete the maxillary alveolar arch.

In summary, the primary palate, consisting of the hard palate anterior to the incisive foramen, the premaxilla, and four maxillary incisor teeth are all derived from the frontal nasal process. The secondary palate, consisting of both the hard and soft palate, are derived from the palatal processes. (See Figs. 2.17 and 2.18.)

Derivatives of Branchial Arches 2–5

The second branchial arch also has a cartilaginous precursor, Reichert's cartilage. Reichert's cartilage lies dorsal to the otic capsule and is responsible for the development of the stapes (except its foot plate) and the styloid process, which combines with the temporal bone. Additional portions of the cartilage and its sheath are converted into the stylo-hyoid body.

The third branchial arches contain cartilaginous precursors that ossify to form the greater horns of the hyoid bone and caudal portion of the hyoid body.

The fourth and fifth branchial arches remain cartilaginous and do not ossify. The fourth arch differentiates into the cuneiform and thyroid cartilages, while the fifth arch transforms into the corniculate, arythenoid and cricoid cartilages (see Fig. 2.19).

The Pharyngeal (Branchial) Pouches and Their Derivates

Toward the end of the fourth gestational week, five sets of paired endodermal sacs, branchial pouches, invaginate the lateral walls of the primitive pharynx. Corresponding ectodermal branchial grooves deepen, pushing aside intervening mesenchyme until the grooves come into contact with their corresponding pouches. The endoderm of the pouch briefly contacts the ectoderm of the groove, producing a so-called closing plate of the tissue. Occasionally these plates become perforate in humans, resulting in branchial cleft cysts and tracts. At this stage of human development the embryo has a homologous structure to the functional branchial clefts of fish and tailed amphibia, a transient demonstration of our ancestral predecessors.

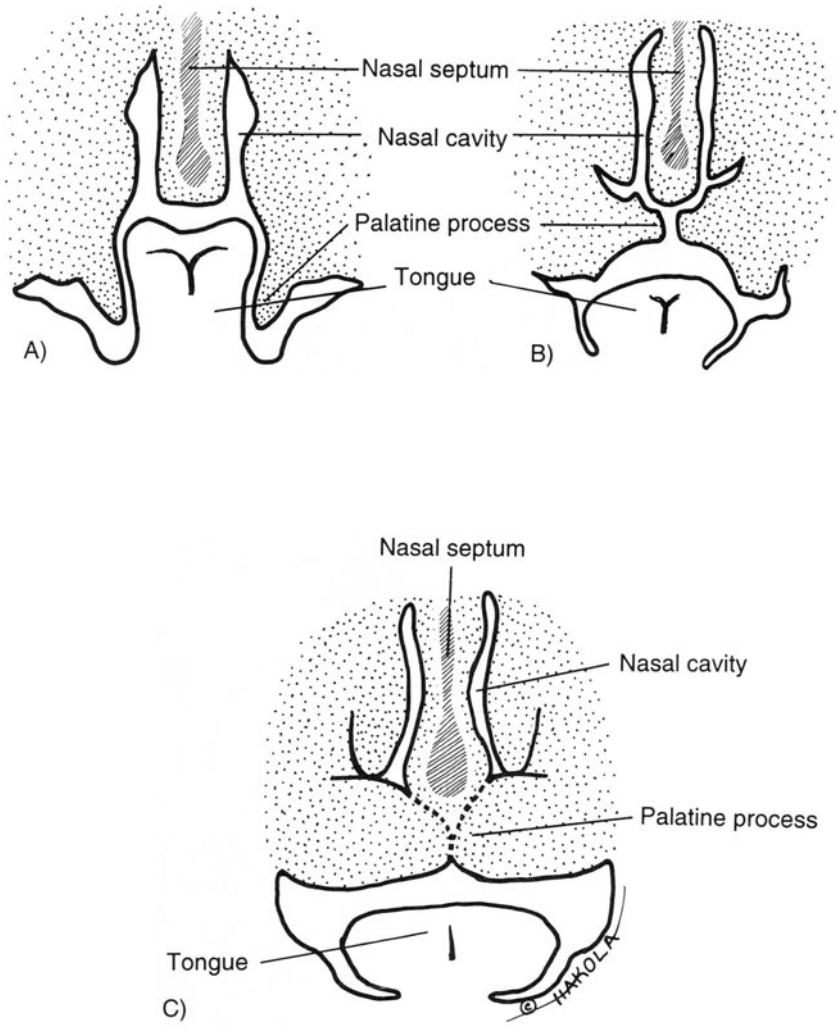

Figure 2.17. (A) Frontal section through the head of a human embryo of 24 mm (8 weeks). The tongue is high and narrow between the vertical palatine processes. (B) Frontal section through the head of an embryo of 30 mm (9 weeks). The tongue has descended below the palatine processes and lies flat and wide within the mandibular arch. The palatine processes have now assumed a horizontal position. (C) Frontal section at about 10 weeks. The horizontal palatine processes have now fused with each other and the nasal septum.

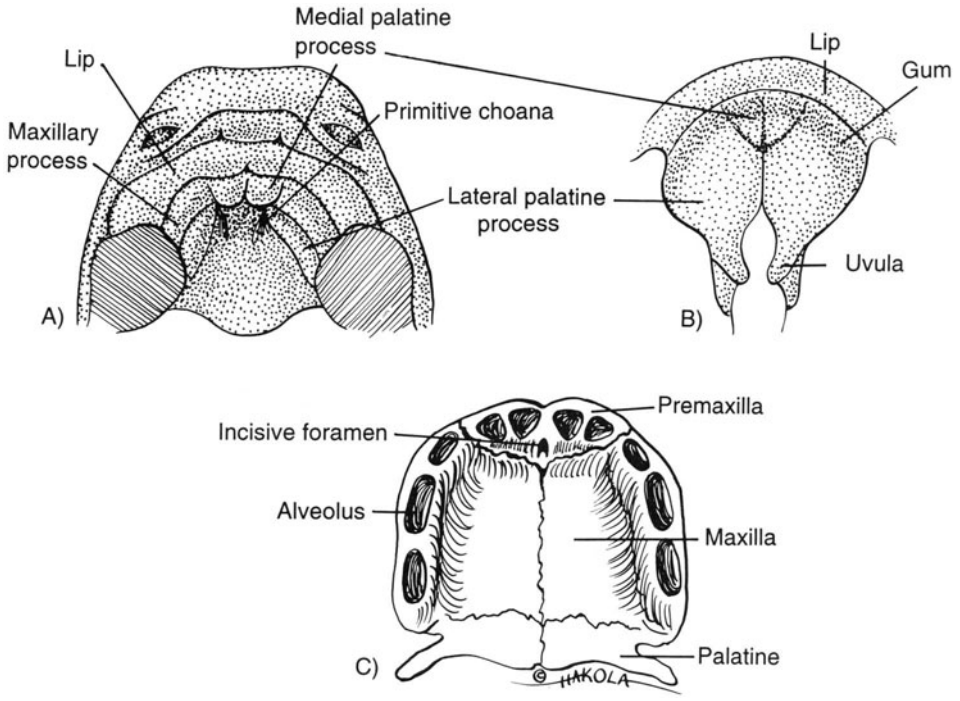

Figure 2.18. Development of the human palate viewed from below. (A) At 8 weeks, (B) at 9 weeks with the beginning of fusion of the palate from anterior to posterior. (C) The hard palate as shown in the skull of a 9-month-old infant with fused premaxillae, maxillary palatal shelves, and paired palatine bones.

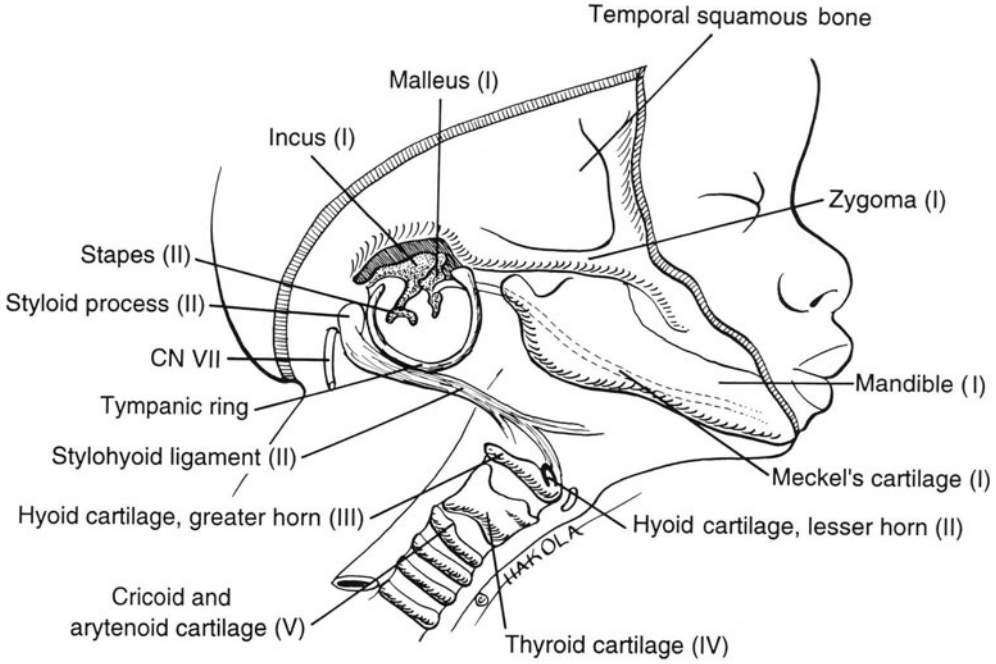

Figure 2.19. Schematic diagram of the fetal head showing the derivatives from the branchial arches I–V.

Only the closing plate of the first branchial pouch remains to form the tympanic membrane. The other pouch closing plates are reseparated by mesenchymal ingrowth.

The first pouch retains its lumen and differentiates into the auditory (eustachian) tube, and tympanic cavity of the middle ear. The second pouch becomes the fossa and covering epithelium of the palatine tonsil. The third, fourth, and fifth pouches give rise to the thymus, parathyroids, and ultimobranchial bodies. (See Fig. 2.20A and 2.20B, and Table 2.1.)

The Oral Glands

True salivary glands are distinctive characteristics of mammals as the only animals that chew properly. They are usually regarded as ectodermal derivatives and have a common plan of origin and development. The primordium arises as an epithelial bud and grows by branching into a bush-like system of solid ducts whose end twigs round out into berry-like secretory acini. Secondary hollowing out of the whole system and specialization of the acinal cells complete the epithelial differentiation. The

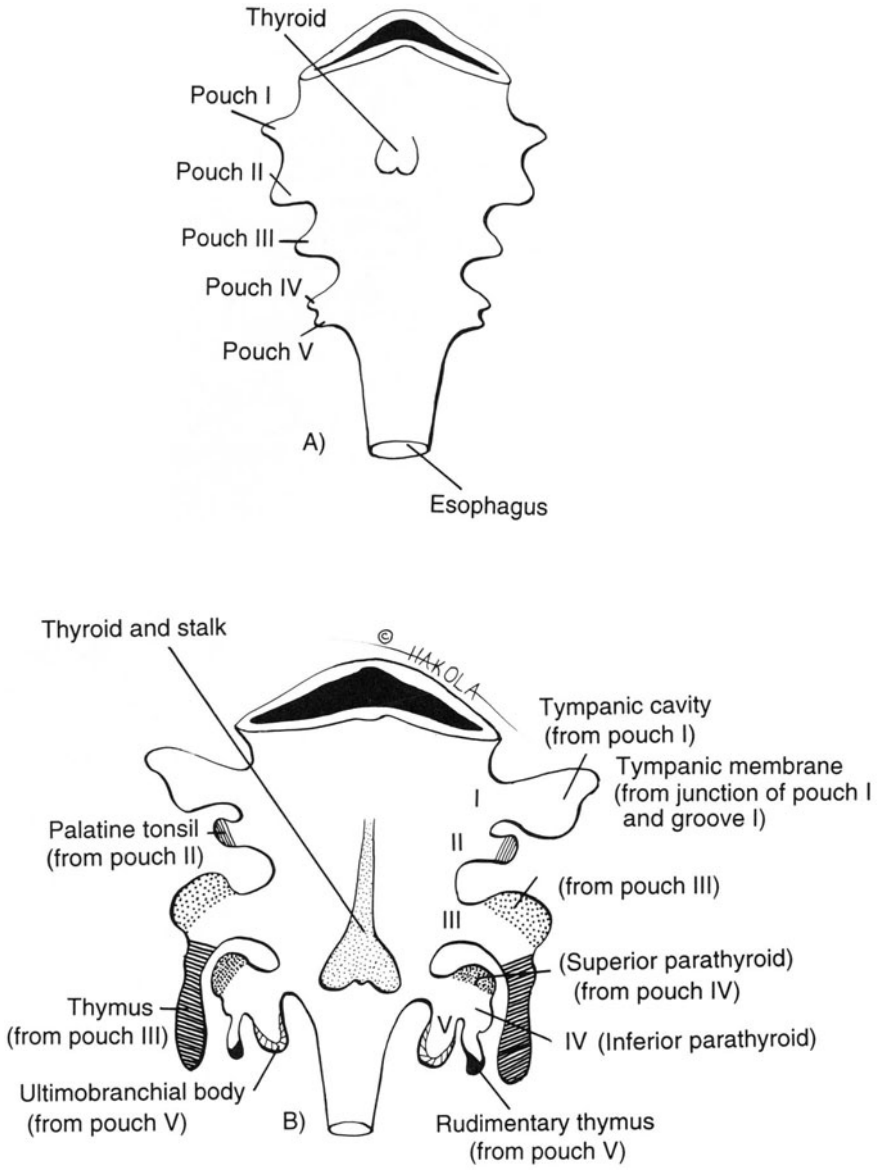

Figure 2.20. (A) The human pharynx with pouches I–V at 4 weeks. (B) The human pharynx with the pouches and their derivatives at 6 weeks. Because of differential migration at 7 to 8 weeks, the pararthyroid derivative from Arch III becomes the inferior parathyroid of the adult, and the parathyroid derivative from Arch IV becomes the superior parathyroid.

Table 2.1. Derivatives of the pharynx and its pouches.

Roof	Level of Pouch I	Level of Pouch II	Level of Pouch III	Level of Pouch IV	Level of Pouch V
Roof	Caudal end of soft palate?	Pharyngeal tonsil. Pharyngeal bursa.			
Sides: Dorsal wing of pouch.	Tympanic cavity: Lining of drum. Mastoid cells. (Rest of pouch: Auditory tube.)	Palatine tonsil: Fossa Epithelium of: Surface. Crypts. (Tonsil develops at site of pouch, but not from it.)	Inferior parathyroid	Superior parathyroid gland.	An aytypical pouch. Derivative: Ultimobranchial body (lateral thyroid).
Sides: Ventral wing of pouch	Obliterates as such.		Thymus: Reticulum. Corpuscles.	Rudimentary thymus in some specimens.	
Floor: (Arch relations.)	Tongue body (I, II). Thyroid gland. Foramen caecum.	Tongue root (II, III). Lingual tonsil.	Epiglottis (III, IV).	Larynx (IV, V). Trachea. Lungs.	

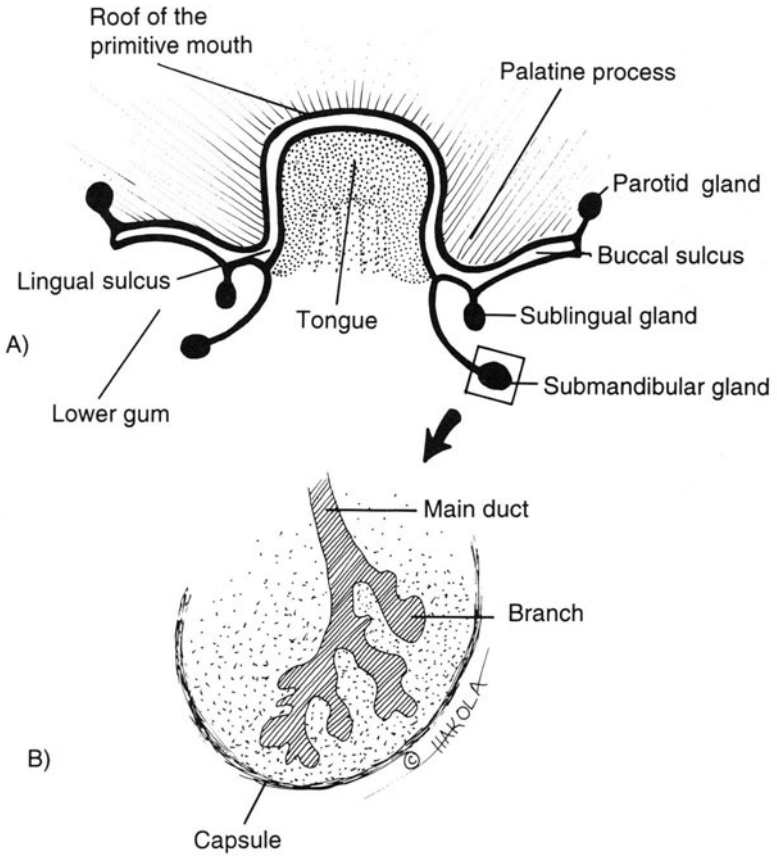

Figure 2.21. (A) Eight-week cross-section through the tongue and developing jaws showing early development of the salivary gland buds. (B) Hollowing out of the solid buds to form the salivary ducts and acini.

dense mass of mesenchyme in which the epithelial primordium lies, furnishes an enveloping capsule and subdivides the gland. By the third month the major salivary glands are organized, and by the sixth month, acinal cells and a canalized duct system are present. Acinal differentiation is not complete until some time after birth even though salivary enzyme appears to be produced by the fetus.

The parotid glands appear first at 6 weeks. The submandibular glands arise at the end of the 6th week. Each sublingual gland appears during the 8th week. The minor salivary glands appear as separate, smaller aggregates at 3 months from multiple epithelial buds in the labial, buccal, and palatine submucosa (see Fig. 2.21).

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CHAPTER 3

Anatomy of the Head and Neck

Andrew Wexler

Successful maxillofacial surgery requires an intimate knowledge of static and functional anatomy of the head and neck. This chapter will provide a brief review of these areas that are of greatest importance to the maxillofacial surgeon. This chapter is not intended to be a detailed or all-encompassing view of head and neck anatomy, for which a standard textbook of anatomy is the best reference. Our approach to the head and neck is that encountered by a scalpel, beginning with the most superficial aspects of the anatomy and delving deeper, providing a plane-by-plane description of the important structures. Areas of particular interest, such as the orbit and submandibular region, will be highlighted separately.

Facial Innervation: The Trigeminal Nerve

The skin of the face and scalp derives its sensation from the terminal branches of Cranial Nerve V, the trigeminal nerve. The trigeminal nerve has three sensory roots that arise from the gasserian ganglion. The three divisions are the ophthalmic (VI), the maxillary (V2), and the mandibular (V3) (Fig. 3.1A and 3.1B).

The ophthalmic division of the nerve exits through the superior orbital fissure after leaving the gasserian ganglion and divides into three major branches: the frontal, the lacrimal, and the nasal ciliary.

The frontal branch of V1 exits at the superior rim of the orbit as the super trochlear nerve, which gives sensation to the conjunctiva, and the skin of the upper lid and the forehead; and the supraorbital nerve, which gives sensation to the upper lid, the forehead, and the scalp. It is important to note that the supraorbital nerve's distribution extends as far posteriorly as the occiput. Accordingly, laceration of this nerve at the level of the forehead or at the superior portion of the orbit will result in hyposthesia of the forehead and the scalp as far back as the occiput.

The lacrimal branch of V1 provides sensation to the conjunctiva and skin of the upper eyelid, and also carries postganglionic parasympathetic fibers from the pterygopalatine ganglion to the lacrimal gland (Fig. 3.2).

The nasal ciliary branch of V1 carries sensation to the nose, to the sclera, and to the cornea of the eye. Its nasal branches provide sensation to the nasal septum in the lateral wall of the nasal cavity, as well as to the skin of the ala, the apex, and the vestibule of the nose. The anterior and posterior ethmoidal branches provide sensation to the ethmoidal and frontal sinuses.

The maxillary division (V2) of the trigeminal nerve exits the cranium through the foramen rotundum and passes through the pterygopalatine fossa, where it gains postganglionic parasympathetic fibers that are carried with the nerve to the nasal and palatine glands. The nerve then branches to form a greater palatine nerve, which supplies the hard palate to the incisor teeth; a lesser palatine nerve, which supplies the soft palate and uvula, as well as the tonsils; and a nasal palatine nerve that runs across the roof of the nose, traverses the nasal septum, and exits through the incisive foramen where it innervates the anterior hard palate and the alveolar margins of the incisors. Additional small branches supply innervation to the superior concha, the septum, and the nasal pharynx.

Of particular interest to the maxillofacial surgeon are the posterior, middle, and anterior superior alveolar nerves. The posterior superior alveolar nerves supply sensation to the first, second, and third molars. The middle superior alveolar nerve supplies sensation to the upper molars, and the anterior superior alveolar nerve supplies sensation to the upper incisors and canine teeth (Fig. 3.3). The maxillary branch then courses along the floor of the orbit, exiting the maxilla below the infraorbital rim, where, as the infraorbital nerve, it then provides sensation to the skin of the lower eyelid, the anterior part of the cheek, the malar prominence, the upper lip, and the anterior part of the temple. Fractures of the inferior floor of the orbit frequently transverse the infraorbital canal, which results in hypoesthesia along

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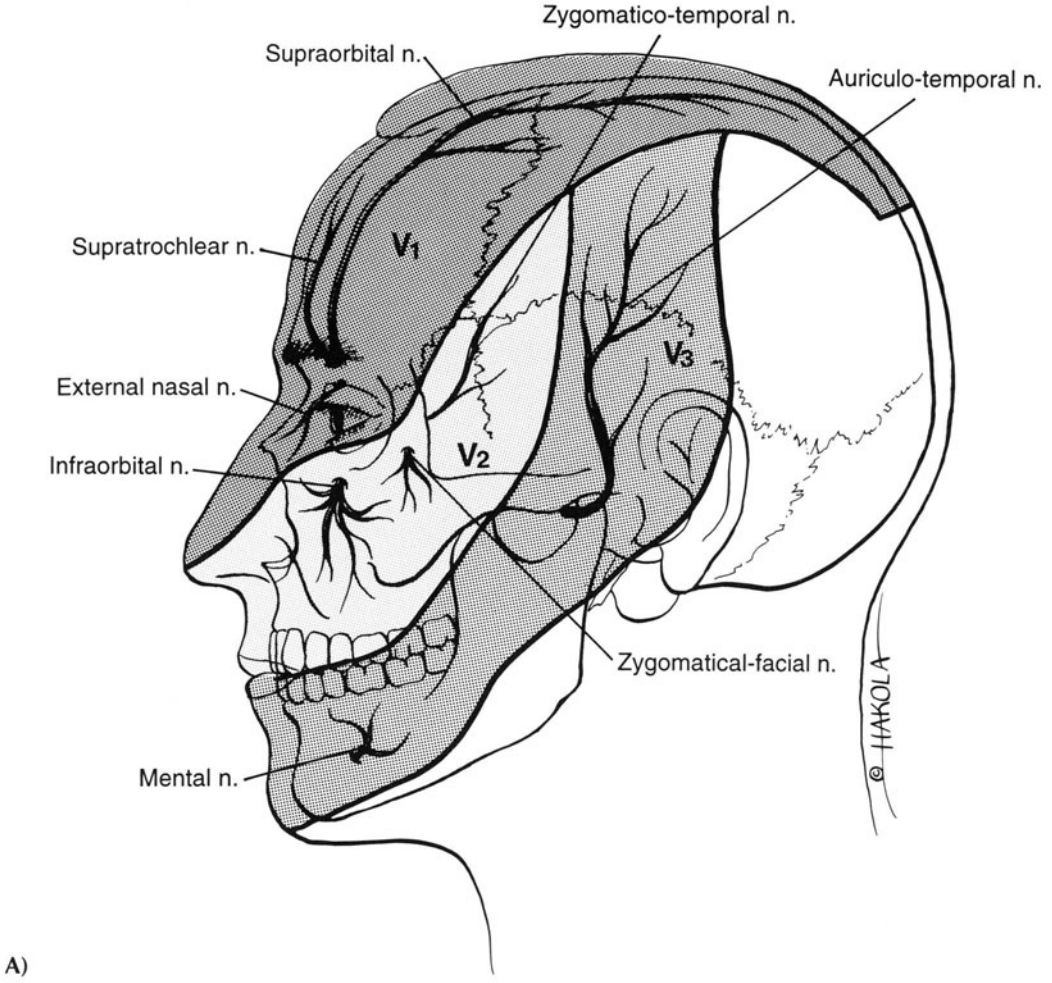


FIGURE 3.1. (A) Superficial sensory dermatomes from the terminal branches of the trigeminal nerve.

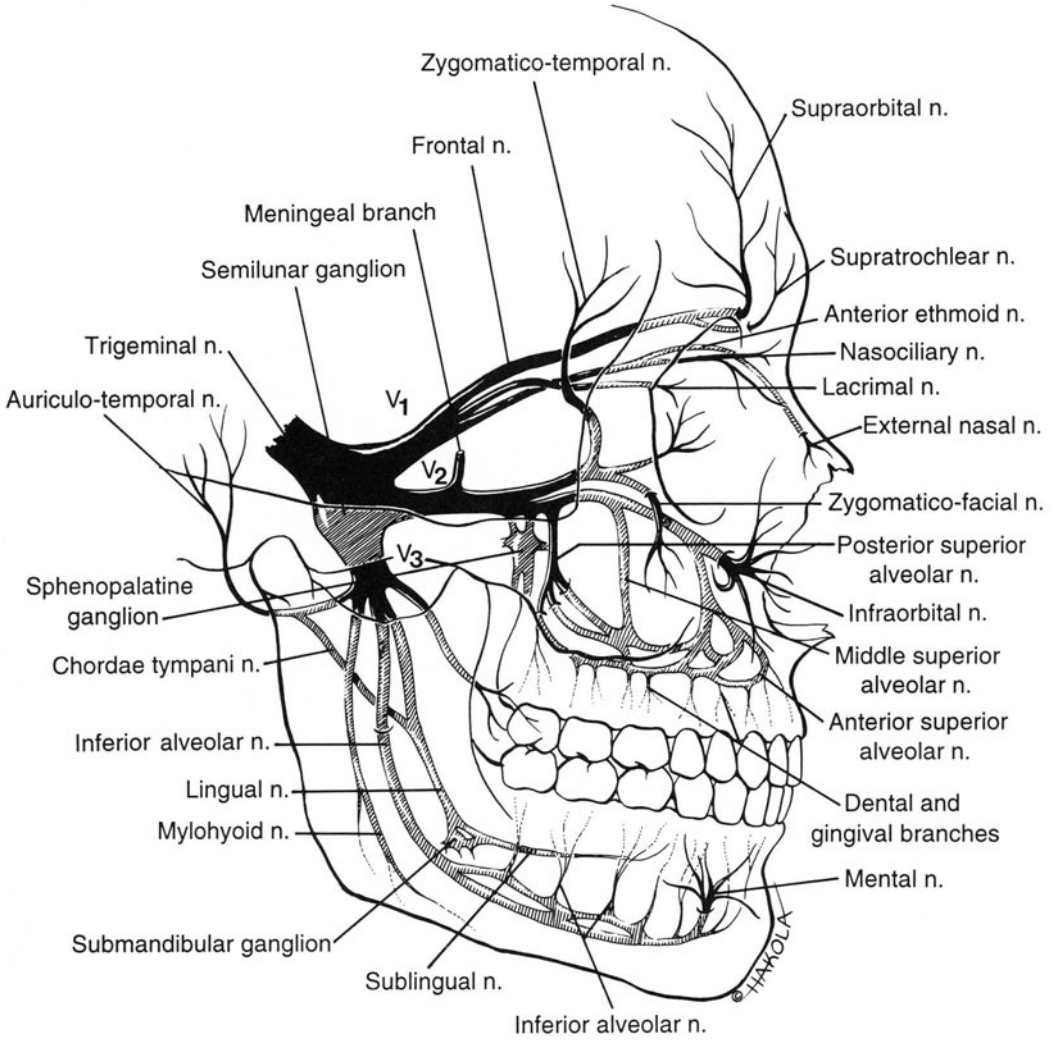


Figure 3.1. *Continued* (B) The trigeminal nerve (Cranial Nerve V).

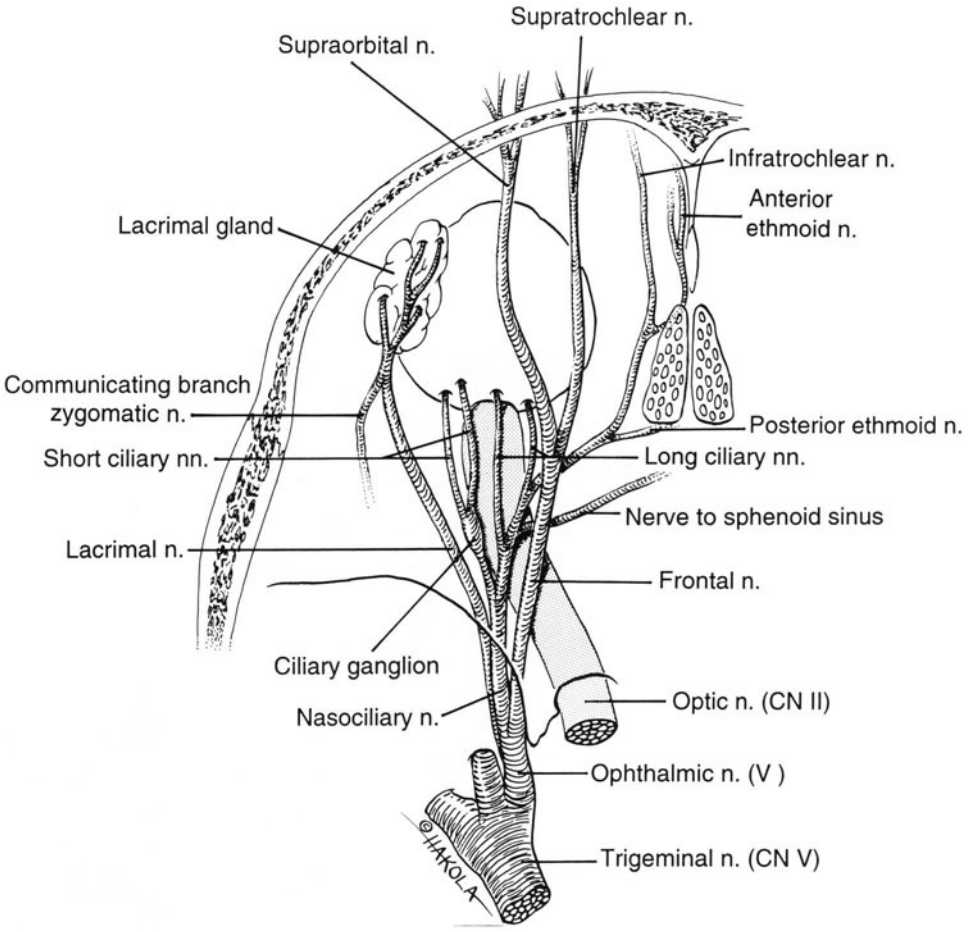


Figure 3.2. Course and branches of the ophthalmic nerve in the orbit.

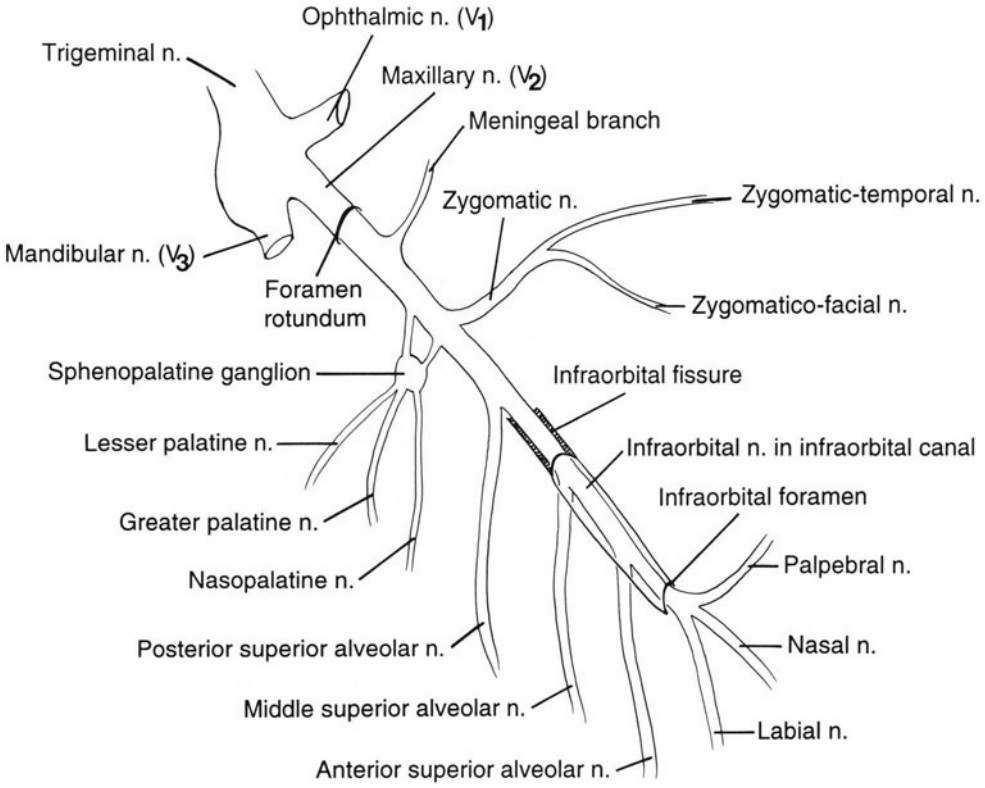


Figure 3.3. Schematic diagram of the maxillary nerve (V_2).

the infraorbital nerve distribution. Accordingly, patients who present after trauma with decreased sensation of the upper lip and anterior cheek should be suspected of having a fracture of the floor of the orbit.

The mandibular division of the trigeminal nerve exits the cranium through the foramen ovale, where its sensory branches then pass through the semilunar ganglion (Figure 3.4). These sensory branches are then joined by a motor branch and together they form the main trunk of the mandibular nerve. The main trunk gives off three motor branches to the medial pterygoid, the tensor tympani, and the tensor palatini muscles. The nerve then divides into an anterior and a posterior division. The anterior division provides motor innervation to the muscles of mastication as well as one sensory branch, the long buccal nerve, which is responsible for sensation to the buccal mucosa and lower gums. The posterior division, as it passes inferiorly in line with the ramus of the mandible, gives off an auriculotemporal nerve that provides sensation to the temporomandibular joint, the skin over the temporal region of the ear, the lateral aspect of the scalp, and the tympanic membrane. Prior to entering the inferior alveolar canal on the medial side of the mandible, the posterior division of the mandibular nerve gives off the lingual nerve, which courses posterior to the retromolar trigone and enters the tongue to provide general sensation to the anterior two thirds of the tongue, the floor of the mouth, and the gums. Prior to entering the tongue, the lingual nerve receives the chorda tympani from the facial nerve, a branch of the seventh cranial nerve that carries taste fibers from the anterior two thirds of the tongue. The chorda tympani also carries autonomic parasympathetic nerves to the submandibular and sublingual glands. After giving off the lingual nerve, the mandibular nerve then enters the inferior alveolar foramen and is now designated the

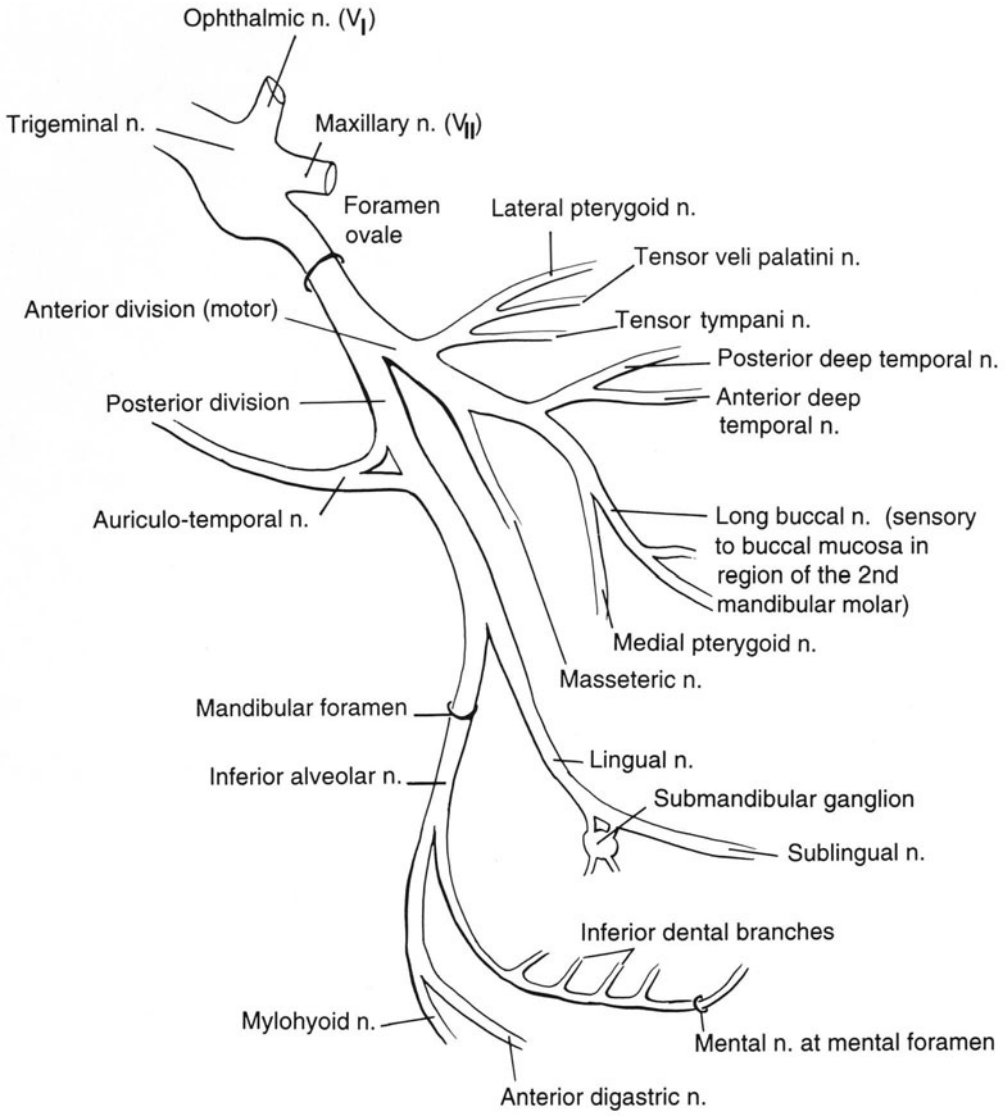


FIGURE 3.4. Schematic diagram of the mandibular nerve (V_3).

inferior alveolar nerve. The inferior alveolar nerve runs within the inferior alveolar canal to the substance of the body of the mandible. The inferior alveolar nerve gives off motor branches to the mylohyoid muscle and the anterior belly of the digastric muscle, as well as sensory branches to the premolar and molar teeth of the mandible. The terminal sensory branch of the nerve exits the mandible through the mental foramen where it provides sensation to the lower lip and chin.

Fractures of the mandible that transverse the inferior alveolar canal are frequently associated with decreased sensation in the chin and lip on the involved side. It is the alveolar nerve that is of greatest risk during a sagittal split osteotomy of the mandible.

The diverse interconnections of the trigeminal nerve throughout its facial distribution are responsible for a multitude of referred pain syndromes. A common example of these would be a sinusitis of the maxillary sinus being referred through the maxillary sensory branches to the pharyngeal nerve and being perceived as a pain in the back of the throat. Similarly, pain from dental caries of the mandible perceived through the inferior alveolar nerve, or a carcinoma of the tongue, perceived through the lingual nerve, might be referred through the mandibular branch of the trigeminal nerve to the auriculotemporal nerve and be perceived as pain in the tympanic membrane.

Lymphatics

The lymphatic drainage of the head and neck (Fig. 3.5) lie in five major groups of nodes, which encircle the head from the occiput to the submental region. Proceeding from posterior to anterior these major groups are the

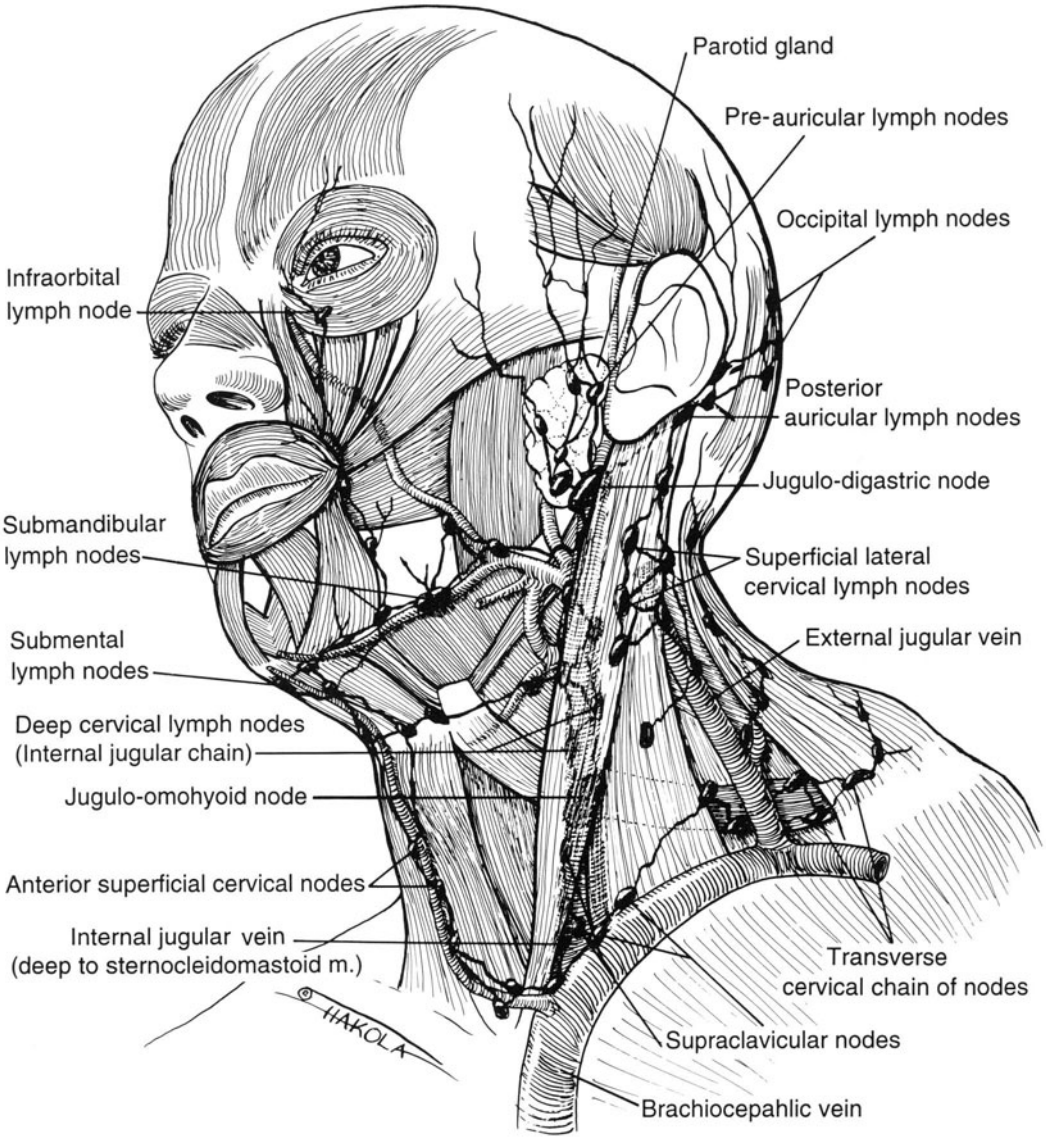


FIGURE 3.5. Lymphatic drainage and principle lymph node groups of the head and neck.

occipital group, a posterior auricular group overlying the mastoid area, a preauricular group lying both superficially and deep to the parotid, a submandibular group lying inferior and posterior to the mandible, and a submental group anterior and inferior to the mandible.

The relationship of the parotid gland to the preauricular nodal group may make parotidectomy a necessary adjuvant in the resection of certain facial and scalp carcinomas. The occipital and posterior auricular lymph node groups drain primarily into the superficial cervical glands surrounding the external jugular vein lying on the sternomastoid muscle. The superficial cervical glands, in turn, drain to the external jugular glands or deep to the anterior group of the cervical nodes that surround the internal jugular veins. A superior deep cervical group of nodes drains the preauricular, submandibular, and submental lymph nodes although they may also drain directly into the inferior deep cervical group. The inferior deep cervical group also receives drainage from the axilla and pectoral regions, as well as the pharynx, the upper part of the thyroid gland, and the larynx.

The Musculofascial Collars of the Neck

Directly beneath the skin and subcutaneous tissue of the neck lies the platysma, a broad flat muscle that originates from the superficial fascia of the thorax, just inferior to the clavicle, and inserts into the outer body of the mandible at its inferior border. The platysma is considered a muscle of facial expression and accordingly is innervated by a branch of the facial nerve, the cervical branch. The muscle fibers of the platysma have been noted to blend with the orbicularis oris, enhancing the depressor function of the lower lip. Directly beneath the platysma anteriorly lies the anterior jugular veins and laterally the external jugular veins. Dissections in this subplatysmal plane, frequently found in face-lift procedures, should avoid these vessels so as not to incur hematoma. Deep to the level of the platysma, the outer muscle collar encircles the neck (Figs. 3.6 and 3.7). This musculofacial collar is composed of the sternomastoid muscle and the trapezius muscle, along with the dense fascia, which both covers and runs between them. The sternomastoid muscle is composed of two separate heads: the round sternal head originating from the superior portion of the front of the manubrium, and a flat clavicular head, originating from the upper surface of the medial third of the clavicle. The two heads of the muscle insert into the outer surface of the mastoid process of the temporal bone, and the lateral part of the superior nuchal line. The muscle is innervated by the XIth cranial nerve, the accessory nerve, along with branches from Cervical Nerves 2 and 3. Normal function of the sternomastoid muscle is to flex the head and the neck and assist in the rotation of the skull. Traumatic injury

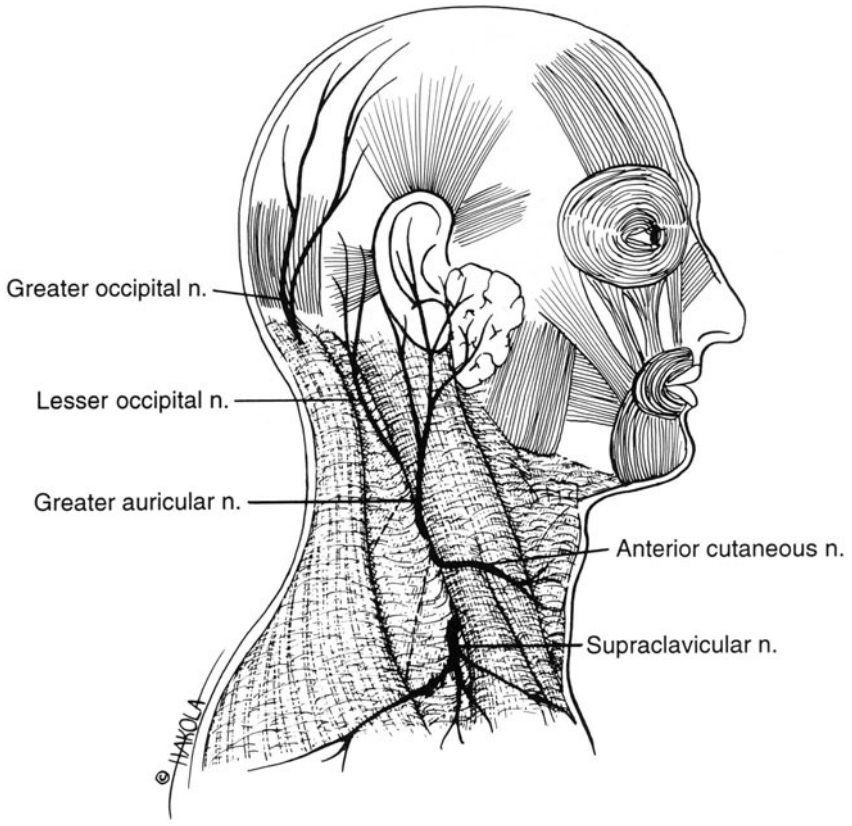


Figure 3.6. Superficial layer of the deep cervical fascia investing the superficial muscles of the neck. Also shown are the superficial cutaneous nerves of the neck and lower ear.

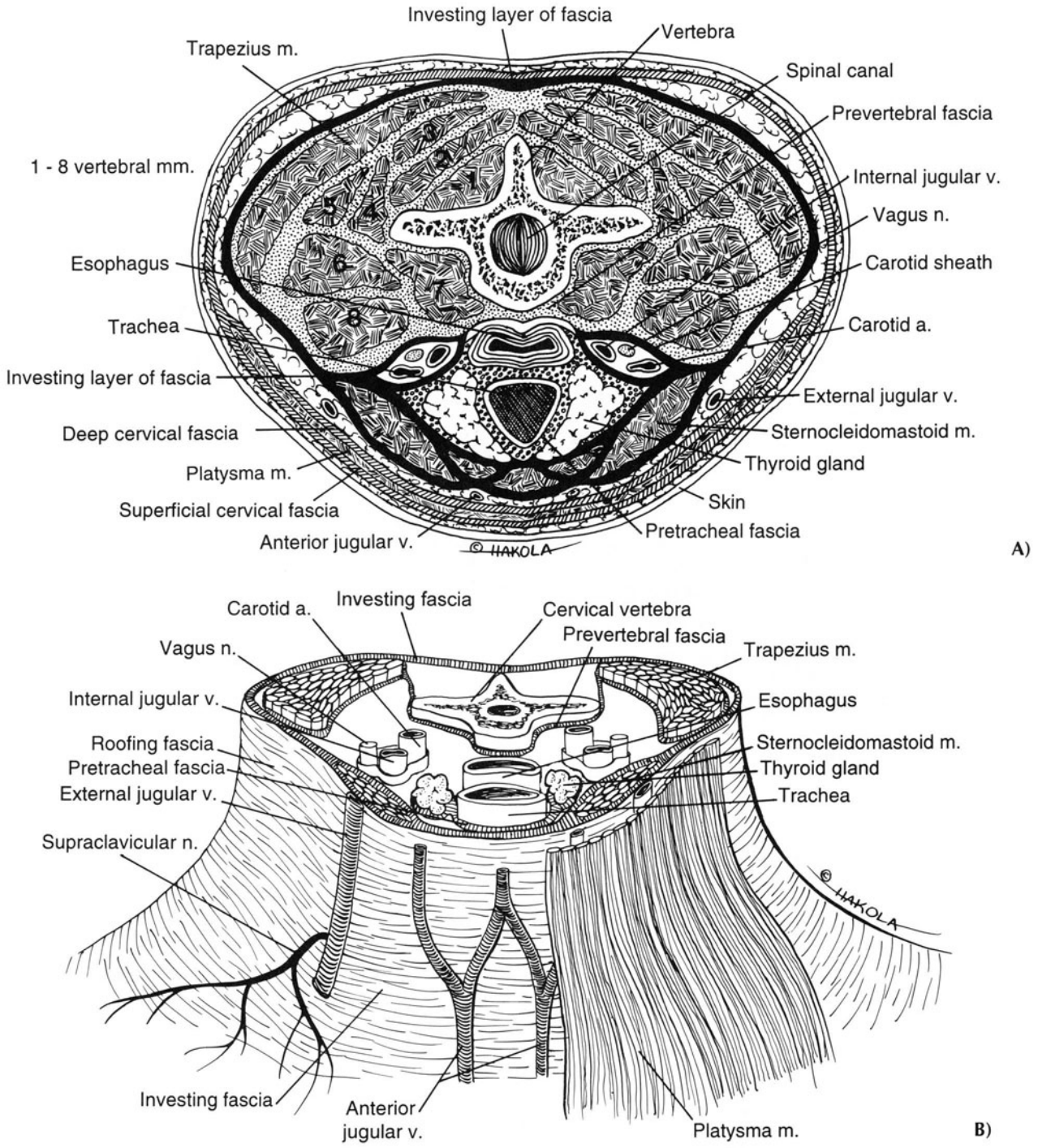


Figure 3.7. (A) The cervical fascial layers. Platysma muscle and anterior jugular veins shown superficial to the deep cervical fascia. External jugular vein shown superficial to investing fascia, which also encloses the sternocleidomastoid muscle. The carotid sheath extends off the investing layer of fascia. The pretracheal fascia surrounds the thyroid gland. Prevertebral fascia envelopes the vertebral body, vertebral, and trapezius muscles. (B) Platysma muscle and anterior jugular veins shown superificial to deep cervical fascia with external jugular vein superficial to investing fascia of sternocleidomastoid.

of the sternomastoid muscle at birth is not uncommon, with resultant scar tissue formation shortening the muscle. This results in congenital torticollis, also known as a wryneck. The development of an asymmetrical skull, plagiocephally, may result from the pull of the shortening. The sternomastoid muscle anatomically divides the posterior triangle of the neck from the anterior triangle of the neck. The posterior triangle is covered by a dense fascia that originates in the prevertebral area and sweeps forward, splitting anteriorly and posteriorly to enclose both the trapezius and the sternomastoid muscle. The fascia, known as the roofing fascia of the posterior triangle, is renamed the investing layer of the deep cervical fascia when it splits to surround the sternomastoid. Anterior to the sternomastoid, the fascia provides separate investments for the strap muscles, the trachea, and the thyroid glands as the pretracheal fascia. The superficial portion of the deep cervical fascia, which passes as the superficial investing layer of the sternomastoid, extends inferiorly from the scapula, clavicle, first rib, and manubrium to the superior nuchal line, the mastoid process, and lower border of the body of the mandible. This fascia then becomes contiguous with the superficial musculoaponeurotic system (SMAS), which extends upward from the border of the mandible to the zygomatic arch. In doing so it creates separate fascia pockets for the parotid and submandibular glands. Accordingly, the parotid gland cannot be said to have a capsule, as it is only the superficial portion of the gland that is covered by this fascia. This same layer of fascia that is briefly broken by the zygomatic arch is seen to continue superior to the arch as the superficial layer of the temporal fascia, also known as the temporal parietal fascia. We may therefore consider the SMAS, and in fact

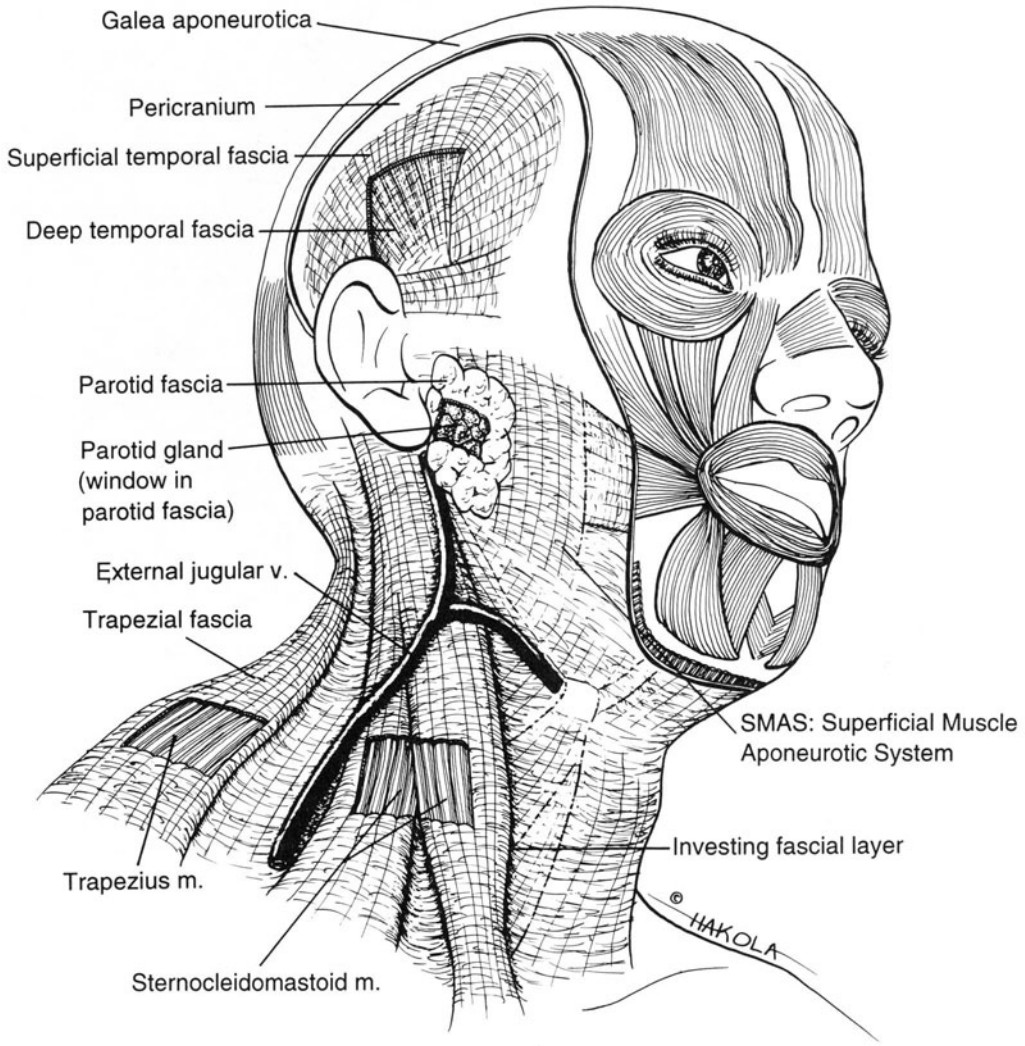


FIGURE 3.8. The investing superficial layer of deep cervical fascia of the head and neck.

the superficial temporal fascia, as being extensions of the superficial portion of the deep cervical fascia (Fig. 3.8). It has been noted that this fascia is pierced anteriorly by the anterior jugular vein and laterally by the external jugular vein. In addition, four sets of nerves pierce the fascia on the lateral aspect of the neck, the lesser occipital nerve, running to the scalp, behind the ear, along the posterior border of the sternomastoid, the greater auricular nerve, running vertically on the sternomastoid muscle toward the ear, the anterior cutaneous nerve, which courses horizontally forward across the sternomastoid muscle to the anterior triangle, and multiple supraclavicular nerves, providing sensation to the anterior neck. All of these nerves are derived from anterior rami running through the cervical plexus.

The Muscles of the Facial Expression

The muscles of facial expression (Figs. 3.9 and 3.10), which include the platysma, are all innervated by the facial nerve (C.N. VII). These muscles, which are all interconnected, may be divided into four major groups:

1. The scalp and ear;
2. the orbits;
3. the nose;
4. the mouth.

All of these muscles insert into the skin and most have a bony origin, except the orbicularis oris, whose multiple interconnected fibers encircle the mouth. Table 3.1 gives an outline of these muscles.

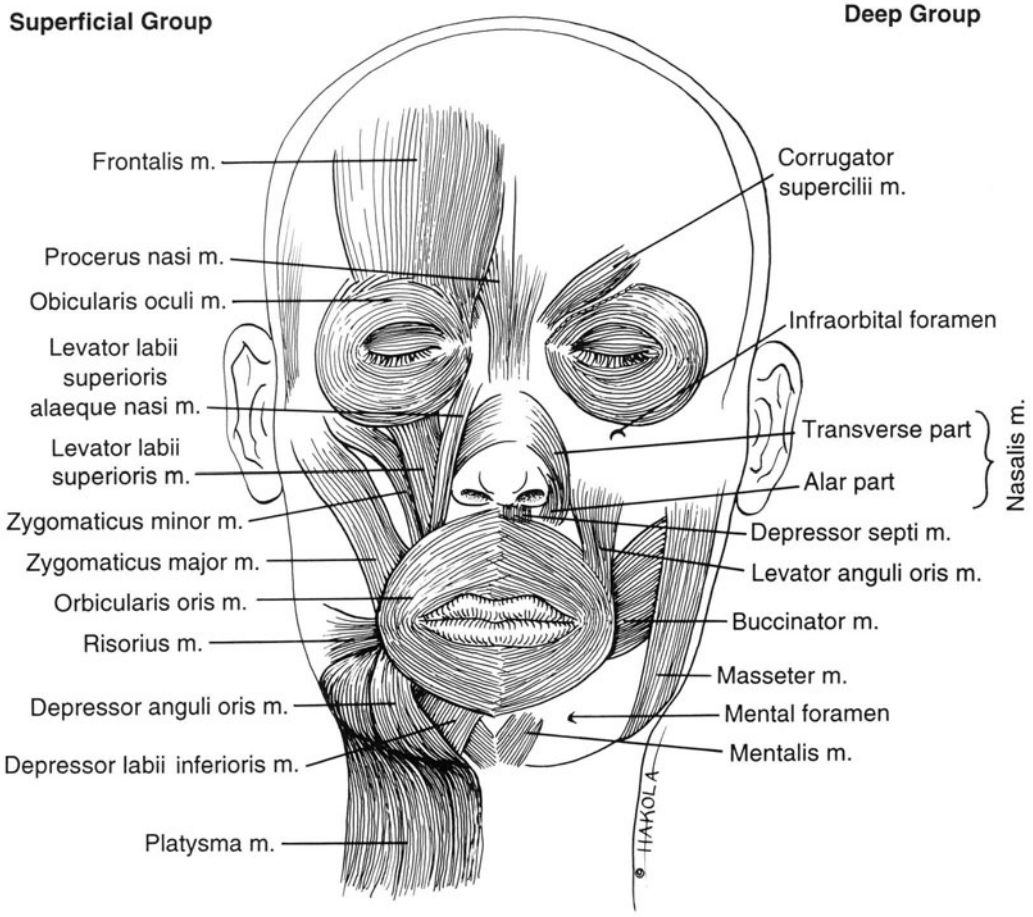


Figure 3.9. Muscles of facial expression. The superficial layer is on the right side of the face; the deep layer is on the left side of the face.

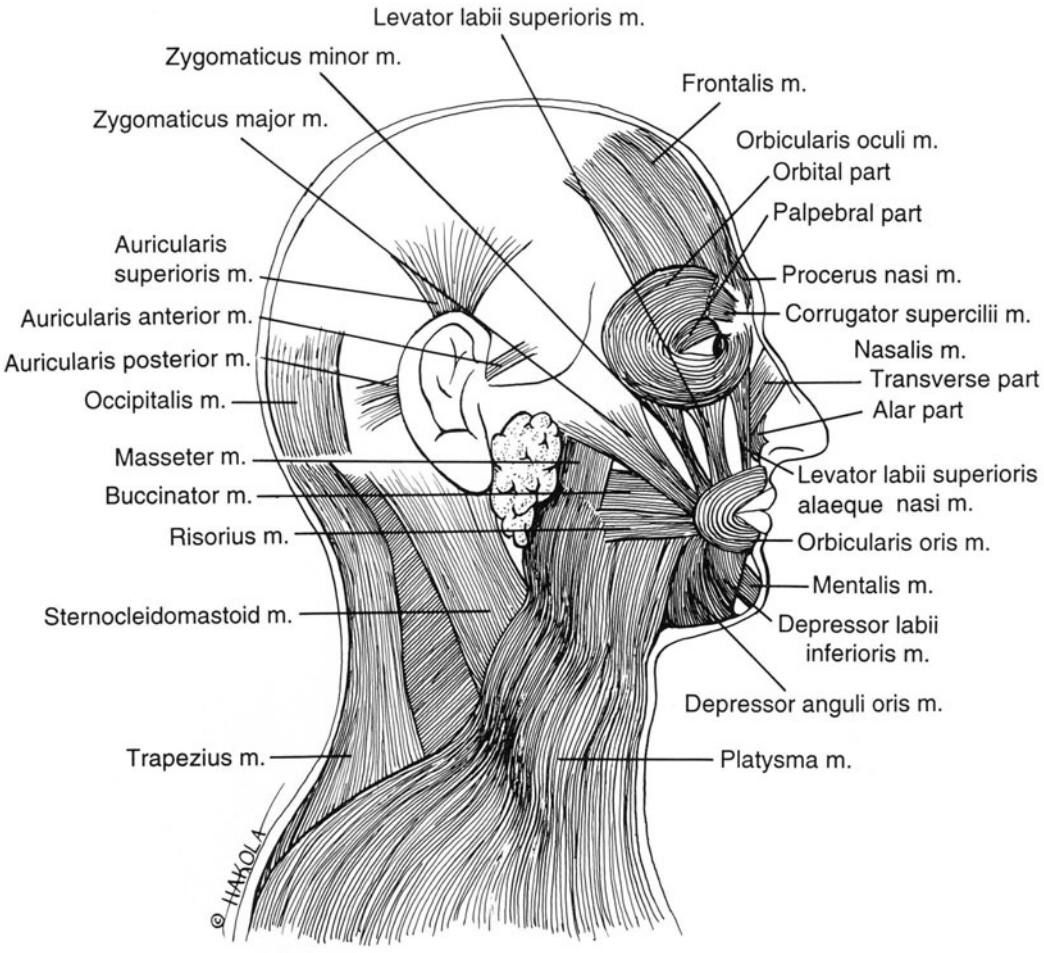


Figure 3.10. Lateral view: Muscles of facial expression.

The Scalp and Paracranium

The soft tissue covering of the skull is composed of hair-bearing skin, subcutaneous tissue, and epicranial aponeurosis, all firmly bound together. Below this lies the periosteum of the skull. The vessels of the scalp are contained in the subcutaneous layer and are adherent to the noncompliant aponeurosis. This aponeurosis prevents effective constricture of the blood vessels when they are cut. This fact allows even minor scalp wounds to bleed more profusely than wounds in other locations. Although bleeding under the aponeurosis can spread over the skull dome, bleeding between the periosteum and the skull is restricted to the individual skull bone to which the periosteum is attached.

The Facial Blood Supply

The facial blood supply is provided by the facial (or external maxillary) artery and drained by the facial vein. The facial artery has its origin on the external carotid artery coursing close to the pharynx. It then runs upward, deep to the posterior digastric muscle, giving off tonsillar branches. The artery loops around the posterior part of the submandibular gland and appears at the inferior border of the mandible where it can be palpated just anterior to the border of the masseter muscle. The ramus mandibularis crosses these vessels superficially at this point. Palpation of these vessels and identification of the nerve's location are important landmarks when making a Risden approach to the mandible. From the border of

Table 3.1. Muscles of facial expression.

Muscle	Origin	Insertion	Facial nerve branch action	
Muscles of the scalp and ears				
frontalis	tendonous galea mastoid portion of temporal bone	skin of forehead, skin of scalp	temporal posterior auricular	moves scalp and forehead
occipitalis				
superior auricularis	galea	auricle	posterior auricular	elevation of ear, draws ear forward and retracts and elevates ear
anterior auricularis	galea	helix	temporal	
posterior auricular	base of mastoid	concha	posterior auricular	
Muscles surrounding the orbit				
orbicularis oculi orbital portion	medial palpebral ligament nasal portion of frontal bone frontal process of maxilla	skin surrounding the eyes	zygomatic and temporal	closes eye, depresses forehead, elevates cheek
palpebral portion	medial palpebral ligament posterior lacrimal crest	loose connective tissue below skin	zygomatic and temporal	closes eye, depresses forehead, elevates cheek
procerus	nasal bone	skin root of nose	zygomatic and temporal	transverse wrinkling of the root of the nose
corregator supercili	frontal bone	medial eyebrow	zygomatic and temporal	Draws eyebrows medially. Vertical wrinkling above the root of the nose
Muscles of the nose nasalis				
transverse	maxilla	nasolabial fold greater alar cartilage	zygomatic + buccal	dilates nares
alar	maxilla over lateral incisor		zygomatic + buccal	depresses nares
depressor septi	incisive foramen of maxilla	septum and posterior ala	zygomatic + buccal	narrows nares
Muscles surrounding the mouth				
orbicularis oris	skin of lips and fibers of surrounding muscle	skin of lips and mouth	buccal	sphincteric action
depressor anguli oris	body of mandible below premolar and first molar teeth	corner of mouth skin and obicularis	marginal mandibular	depresses angle of mouth
depressor labii inferioris	anterior mandible	skin and mucosa of lower lip	marginal mandibular	depresses central lip
mentalis	root of lower incisors	downward into skin of chin	marginal mandibular	draws skin of lip upward, forces lower lip against lower teeth and gums
platysma	clavicle and upper portions of thorax	border of jaw superficial to facial muscles and depressor angular oris	cervical	contracts skin of neck, depresses lower lip at corners
risorius	subcutaneous tissue over parotid	mucosa + skin of lateral corner of mouth	buccal	draws corner of mouth laterally and somewhat upward
zygomaticus major	posterior lateral zygoma	skin and mucosa corner of mouth	buccal and zygomatic	draws corner of mouth laterally and upward
zygomaticus minor	zygoma medial to z-major	skin and mucosa corner of mouth	buccal and zygomatic	draws corner of mouth laterally and upward
levator labii superioris	infraorbital margin of maxilla	skin lateral half of lip	buccal and zygomatic	raises upper lip
levator labii superioris alacque nasi	frontal process of maxilla along the nose	skin of and nasal ala of nose and skin of upper lip	buccal and zygomatic	lifts the lip and widens the naris
buccinator	pterygomandibular raphe upper medial mandibular at the ramus body junction and posterior alveolar process of maxilla	orbicularis and mucosa + skin of lips	buccal branches of facial	flattens cheek against teeth, moves food from buccal sulcus onto teeth

the mandible the artery runs superiorly and medially deep to the platysma, risoris, and zygomaticus. The vessel then courses along the side of the nose toward the medial angle of the eye, where it terminates in the angular artery. Along the way, the facial artery gives off a superior and inferior labial artery, which runs deep to the orbicularis and superficial to the submucosa of the inner lip. It is the inferior labial artery that serves as the axial blood supply for the Abbe flap. The artery's submucosal position allows for division of a majority of the lip, the creation of a thin pedicle, and subsequent ease of flap rotation (Figs. 3.11 and 3.12).

The anterior facial vein runs parallel and posterior to the artery. The vein begins as the angular vein at the angle of the nose and eye. The angular vein communicates with the orbital vein, and consequently with the cavernous sinus. Infections of the face, especially around the upper lip and nose, therefore have the potential for intercranial extension and cavernous sinus thrombosis (Fig. 3.12).

The anterior facial vein empties into the common facial vein, and then into the internal jugular vein. The common facial vein also receives the anterior division of the posterior facial vein. The posterior facial vein is formed opposite the temporomandibular joint, where it receives drainage from the scalp, the temple, and the pterygoid plexus. The vein divides into an anterior division, which drains the common facial vein, and a posterior division, which joins with the posterior auricular vein, to drain into the external jugular.

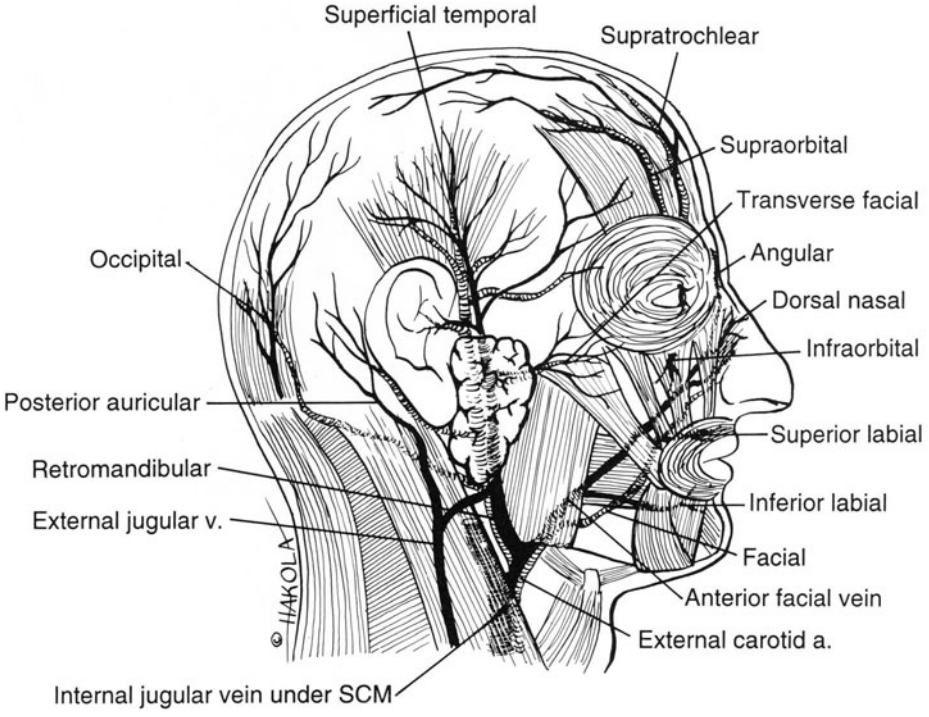


Figure 3.11. Facial vessels lying deep to superficial facial muscles.

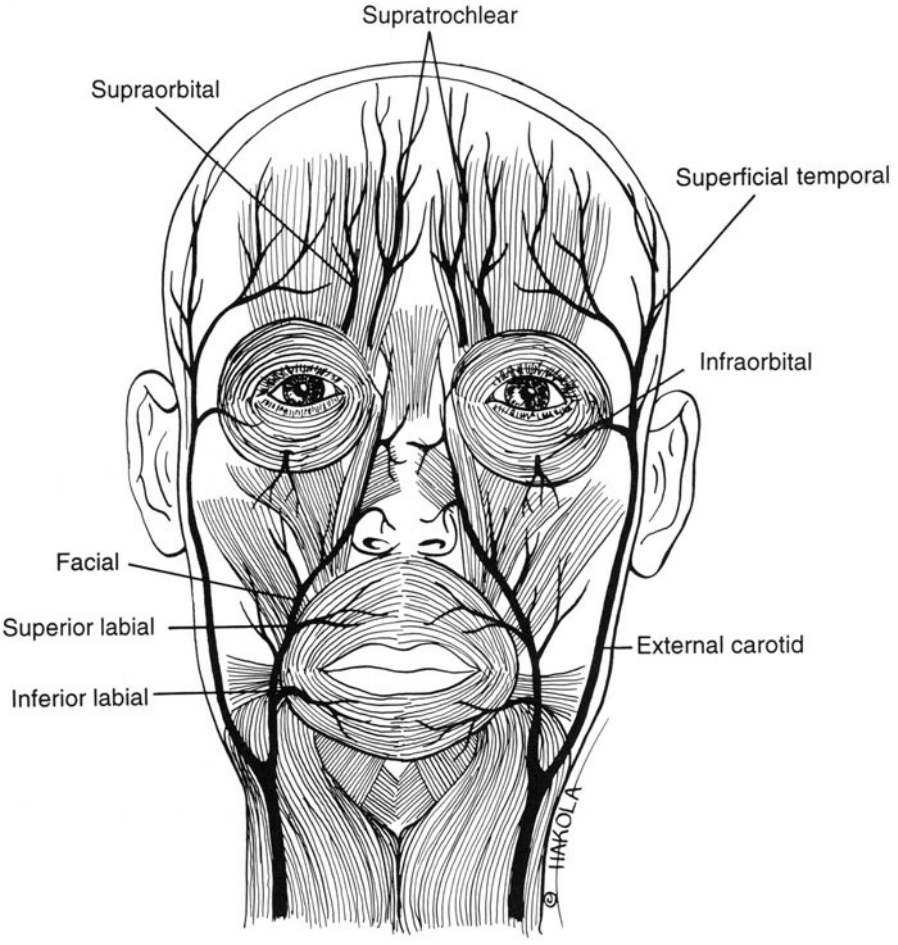


Figure 3.12. Anterior view of facial arteries.

The Parotid Gland

The parotid is the largest of the salivary glands. It wraps itself in a horseshoe-shaped manner around the posterior ramus of the mandible. The deep segment of the gland lies medial to the medial pterygoid along the medial surface of the ramus, while the superficial segment lies lateral to the masseter muscle, along the lateral surface of the ramus. Note that although the parotid has a deep and a superficial segment, it is not divided into two separate lobes, nor does it possess its own fascial capsule (Fig. 3.13). Instead it is covered superficially by an extension of the superficial layer of the deep cervical fascia. At the most inferior portion of the gland, this fascia joins with the fascia running deep to the parotid gland to form the stylomandibular ligament, which separates the parotid from the underlying submandibular gland (Fig. 3.14).

At the anterior edge of the superficial portion of the gland, the parotid (Stensen's) duct may be found exiting the gland and coursing anteriorly across the masseter muscle on a line running roughly between the lobule of the ear and the base of the nasal ala. At the anterior border of the masseter muscle, which lies along a vertical line extending downward from the lateral canthus, the duct turns medially, piercing the buccinator, and enters the mouth through the buccal mucosa at the parotid papilla. The papilla can be found at the level of the junction of the crown and root of the second maxillary molar. Piercing the parotid's substance posteriorly is the main branch of the facial nerve (C.N. VII). The facial nerve trunk exits from the stylomastoid foramen, passes un-

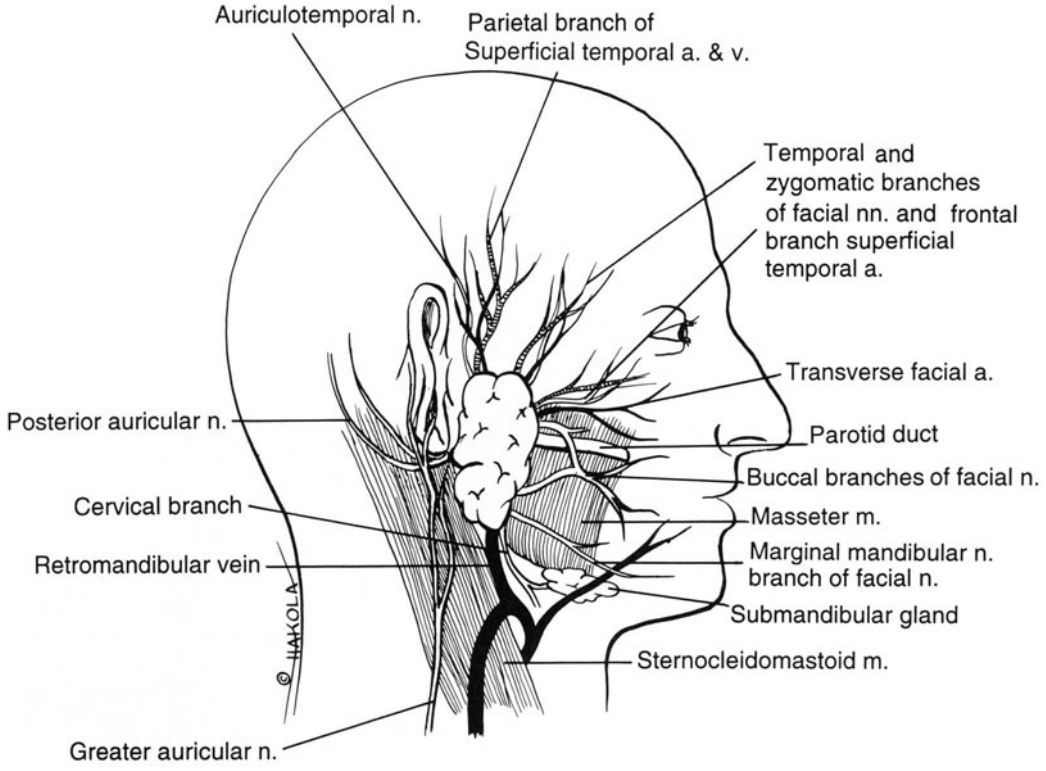


Figure 3.13. Superificial relations of the parotid gland. Anatomy shown with ear rolled forward.

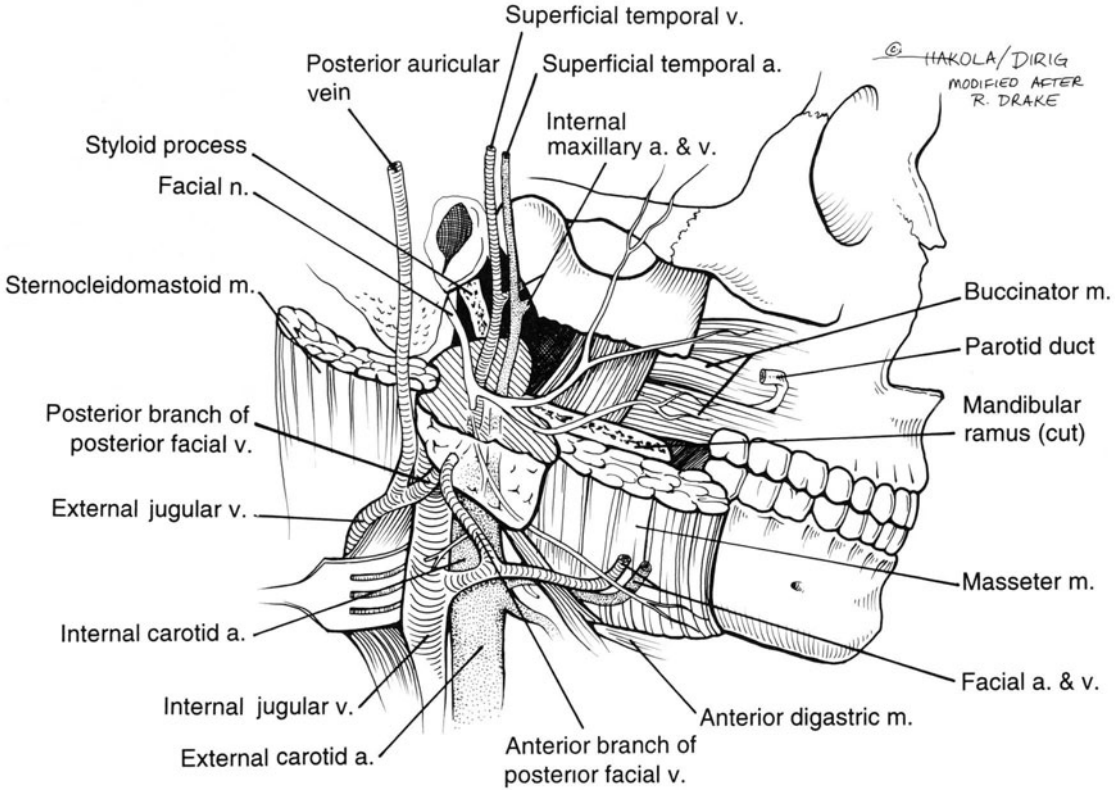


Figure 3.14. Deep relations of the parotid gland.

der the protection of the mastoid process, and then divides within the gland's substance into its five major branches. Arborization of the nerve branches takes place within the gland's substance. Facial lacerations encountered anterior to a vertical line drawn between the lateral canthus and the lateral oral commissure need not be explored for either parotid duct damage or facial nerve damage, as the duct will have already pierced the buccinator muscle and the facial nerve will have sufficiently arborized to an extent that makes repair both difficult and unnecessary (Fig. 3.15). The surgeon should be aware that in a child under the age of 4, the main trunk of the facial nerve runs very superficially in the preauricular region and is not afforded the protection of the mastoid process, which at this stage is not fully developed (Fig. 3.16). Accordingly, lacerations or surgical incisions made in this area risk transsecting the facial nerve trunk.

The parotid's parasympathetic innervation is primarily derived from the glossopharyngeal nerve (C.N. IX) via the auriculotemporal nerve, a branch of the trigeminal nerve (C.N. V). Sympathetic innervation of the gland is via the fibers carried along the adjacent arteries.

The Infratemporal Region

A surgeon engaged in a total parotidectomy will by necessity have entered into the infratemporal region in his or her dissection of the deep portion of the gland. The infratemporal region lies below and medial to the zygomatic arch. The infratemporal fossa is superiorly bordered by the infratemporal crest of the greater wing of the sphenoid. The medial limit is the lateral pterygoid plate. The lateral borders are the ramus and coronoid process of the mandible. Anteriorly, the fossa is bound by the infratemporal surface of the mandible and posteriorly by the posterior border of the mandible. Within the infratemporal region lie most of the muscles of mastication as well as the major blood vessels and nerves to the mouth region.

The mandibular division of the trigeminal nerve (C.N. V) is the main nerve of the infratemporal region

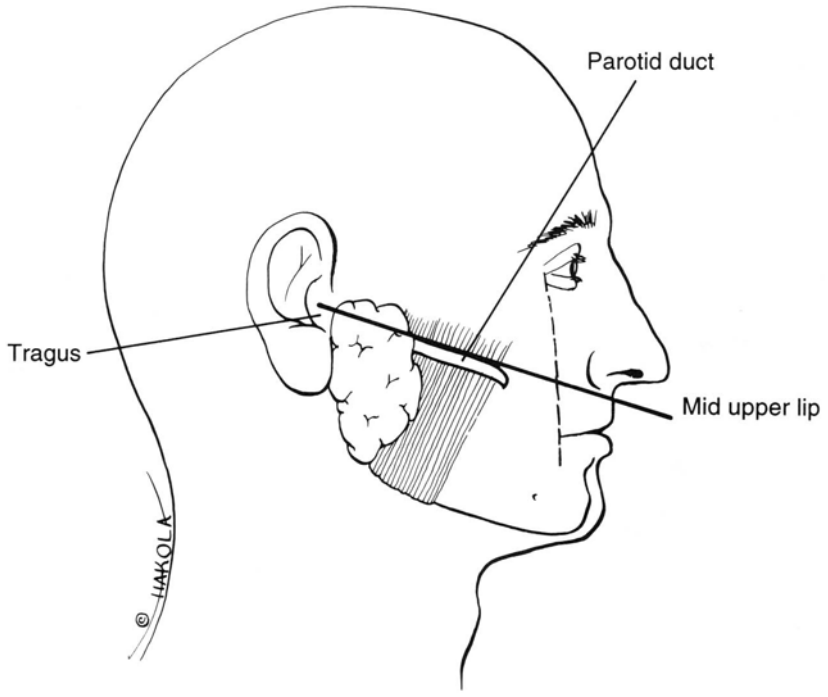


Figure 3.15. The parotid duct is found on a line drawn from the tragus to the mid upper lip. Anterior to the line drawn from the lateral canthus to the lateral commissure, the facial nerve branches have arborized to an extent where repair is not necessary.

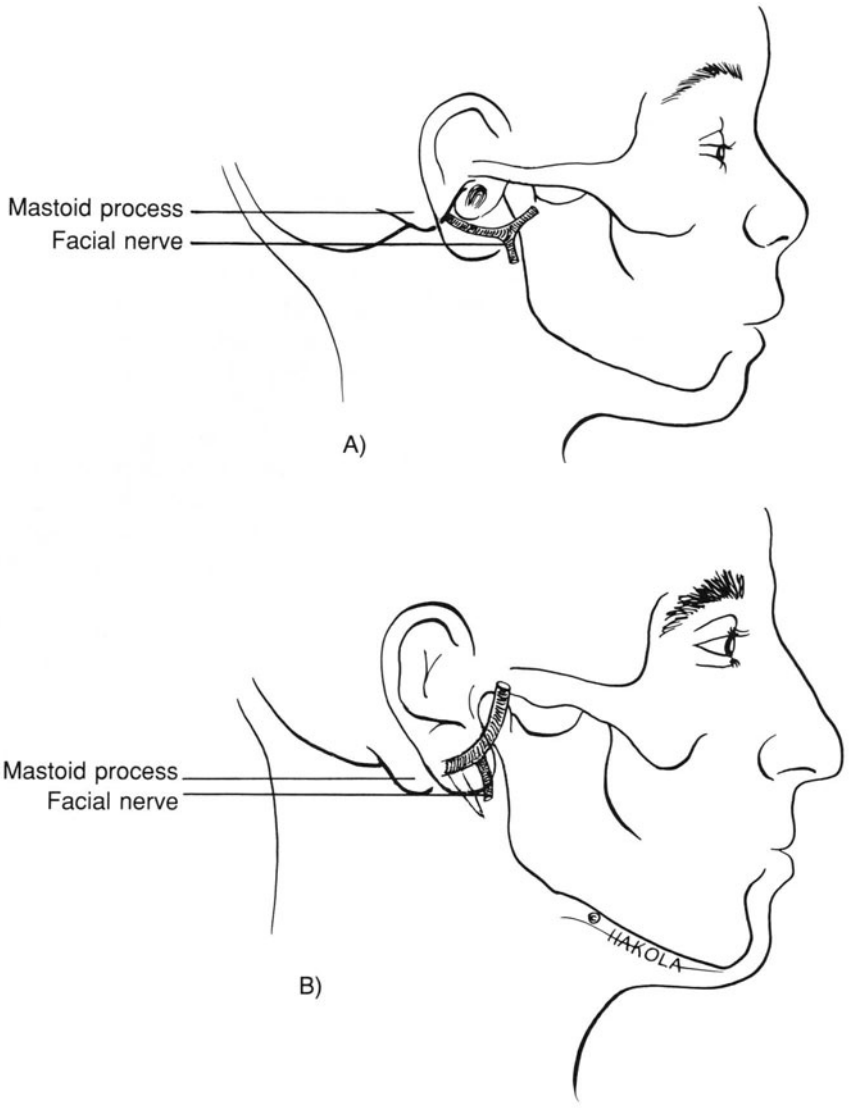


Figure 3.16. Depth of the facial nerve in a child is superficial, whereas the nerve is deeper and protected in the adult by the mastoid process.

(Fig. 3.4). It emerges from the foramen ovale, passing deep to the lateral pterygoid muscle and lateral to the eustachean tube and otic ganglion. The nerve immediately separates into an anterior and posterior trunk. As previously described in the section on the facial nerve, the anterior trunk branches into motor fibers and innervates the pterygoid muscles, masseter, temporalis, and tensor palatini muscles. A single sensory branch, the buccal nerve, passes often with the anterior deep temporal branch between the two heads of the lateral pterygoid. It then runs downward along the medial insertion of the temporalis muscle, pierces the buccal fat pad, and is then distributed to provide sensation to the inner cheek and gingiva.

The posterior trunk of the nerve runs lateral to the tensor and levator palatini. The auriculotemporal nerve, in close proximity to the medial meningeal artery, is the first branch given off from the posterior trunk. The posterior trunk then passes in the preauricular region posteriorly and medial to the temporomandibular joint, and anteriorly to the external acoustic meatus, where it accompanies the superficial temporal artery to supply sensation to the anterior ear, auditory meatus, and the skin of the temple. The posterior trunk of the mandibular nerve then divides into the inferior alveolar and lingual nerves prior to leaving the infratemporal fossa.

The maxillary artery (internal maxillary) is the major vessel of the infratemporal region. The maxillary artery takes its origin from the external carotid artery, which ascends deep to the posterior belly of the digastric and stylohyoid muscles, running approximately parallel to the posterior border of the ramus of the mandible. After

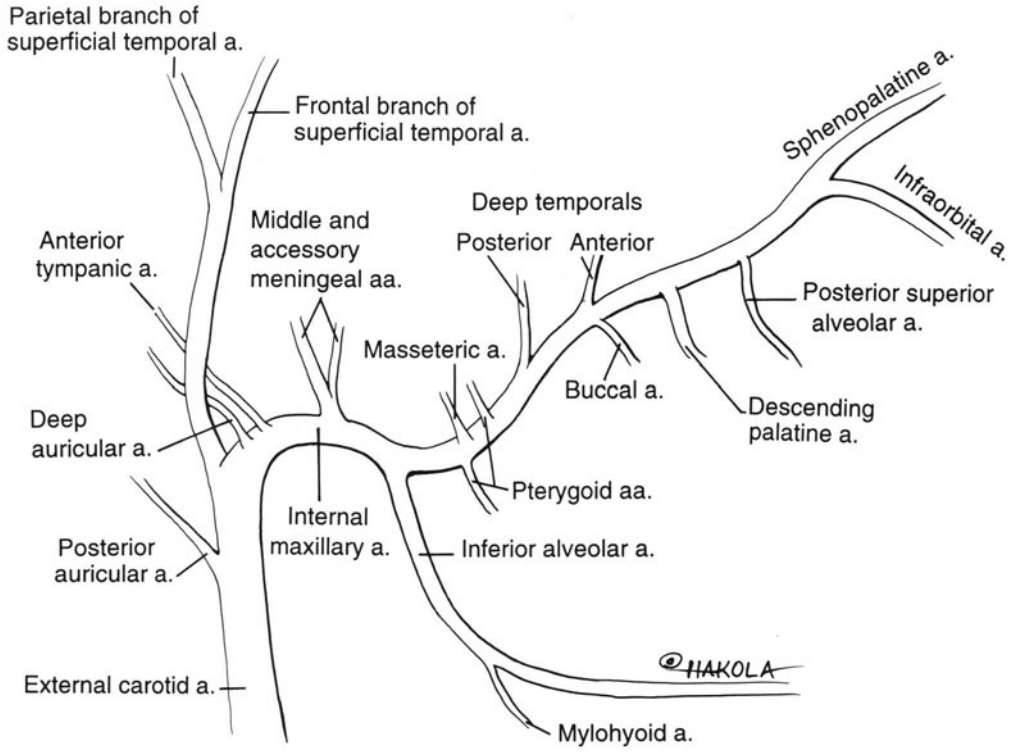


Figure 3.17. Schematic diagram of the maxillary artery and its major branches.

giving off the facial artery (external maxillary) at the level of the angle of the mandible, the external carotid artery divides into its two terminal branches, the maxillary artery and the facial artery. The point of this division is roughly one half to two thirds the distance from the angle of the mandible to the temporomandibular joint, deep to or within the substance of the parotid gland (Fig. 3.17).

The maxillary artery usually passes across the lateral surface of the lateral pterygoid muscle, though it may run deep to the muscle for a short distance and then surface further forward between its two heads. The course of the maxillary artery is anatomically considered in three portions as it traverses the infratemporal region. The first portion is medial to the mandible and posterior to the lateral pterygoid. Four branches arise from this first part of the artery. The deep auricular and anterior tympanic arteries pass upward to supply the external acoustic meatus and the middle ear cavity. The middle meningeal artery, the most important branch clinically, runs upward to enter the skull through the foramen spinosum. The inferior alveolar (dental) artery descends, accompanied by the inferior alveolar nerve into the mandibular canal where it supplies the lower teeth.

The second part of the artery lies in contact with the lateral pterygoid muscle. The branches of this portion of the artery accompany nerves of the same name to the surrounding musculature. The arterial branches are the masseteric, the buccal, two deep temporals, and branches to both pterygoids. The third portion of the artery is that which enters the ptergopalatine fossa in the posterior lateral aspect of the maxilla. The branches of this portion of the artery are the posterior superior alveolar artery to the upper teeth, the infraorbital artery, which enters the infraorbital canal and gives off orbital and superior alveolor branches, the descending palatine artery with its greater and lesser palatine branches, and, last, the sphenopalatine artery, which supplies the nose. Surrounding the lateral pterygoid muscle is a plexus of veins draining the areas supplied by the maxillary artery. The pterygoid plexus drains into the alveolar and posterior facial vein. In maxillofacial trauma it is the sphenopalatine vessel that is most often a source of profuse bleeding. Inability to control the bleeding through reduction of the fracture or nasal packing may require direct exposure and ligation of the third portion of the maxillary artery through a transnasal approach. Failing this, ligation of the external carotid artery may be the only recourse (Fig. 3.18).

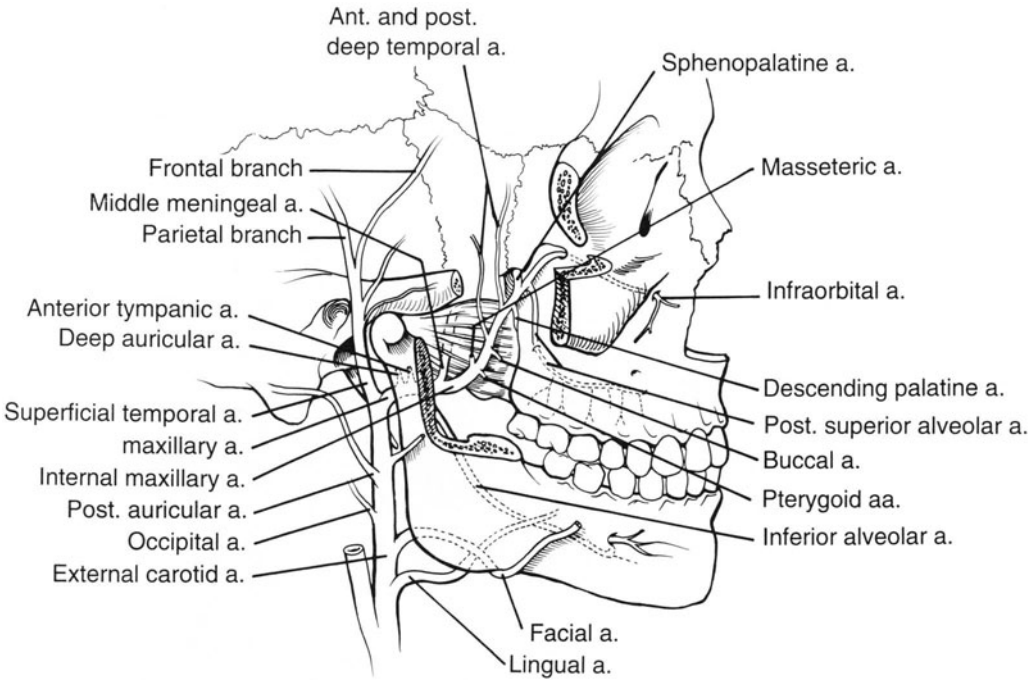


Figure 3.18. The maxillary artery and its branches.

The Muscles of Mastication

The four muscles of mastication all pass through the infratemporal fossa. Their functional anatomy is of primary importance to the maxillofacial surgeon. The masseter muscle originates from two heads. The superficial portion arises from the lower border and the anterior two thirds of the zygomatic arch. The muscle fibers slant downward and backward, overlying the deep head of the muscle to insert into the lateral and inferior portion of the ramus of the mandible. The deep head of the muscle originates from the inner surface of the posterior one third of the zygomatic arch, where it then runs vertically downward to join the insertion of the superficial head at the ramus. The masseter muscles are strong elevators of the mandible. The deep head of the muscle also serves as a mandibular retractor (Fig. 3.19A).

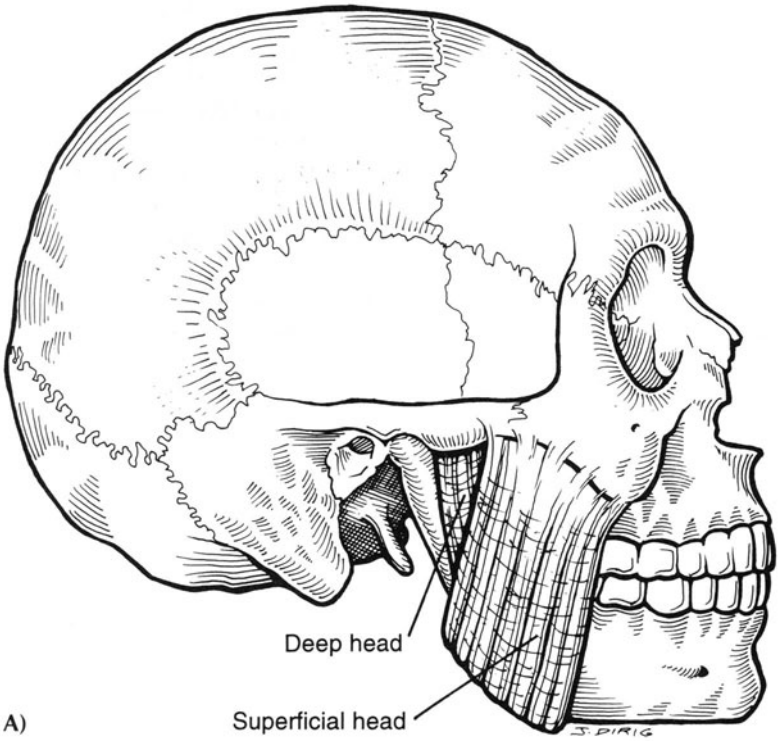


Figure 3.19. (A) The two heads of the masseter muscle.

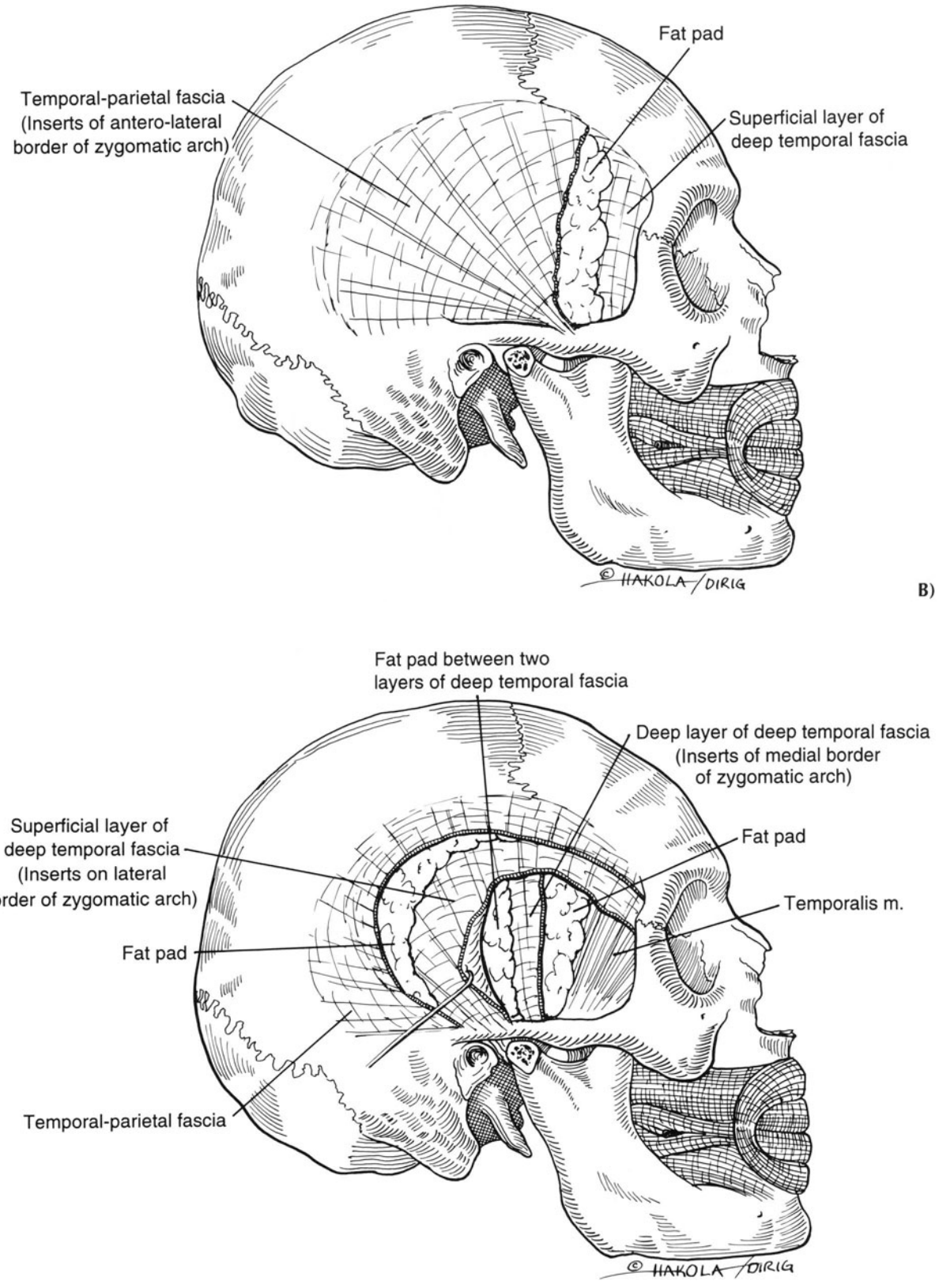


Figure 3.19. *Continued* (B) The temporal-parietal fascia, the most superficial layer of the temporal fasciae. Cutaway shows the fat pad between the temporal-parietal fascial layer and the superficial layer of the deep temporal fascia. (C) The deep temporal fasciae, both superficial and deep layers.

The temporalis muscle originates in the temporal fossa, passes lateral to the maxillary artery and lateral to the pterygoid, and beneath the zygomatic arch, to insert into the coracoid process and the medial side of the ramus of the mandible. The temporalis muscle is covered by two layers of fascia, the temporal parietal (most superficial layer of the fascial layers) and the deep temporal fascia (Fig. 3.19B). The deep temporal fascia is further subdivided into a superficial and a deep layer. The temporal parietal and deep temporal fascia are separated by a layer of loose areolar tissue (Fig. 3.19C). As one approaches the zygomatic arch, the superficial and deep layers of the deep temporal fascia are separated by the superficial temporal fat pad.

The frontal branch of the facial nerve is encompassed by the temporal parietal fascia at 2 finger breaths lateral to the lateral orbital rim & 2 finger breaths above the zygomatic. The temporal branch of the facial nerve is also encompassed by the temporal parietal fascia at the level of the arch. When performing a coronal approach to the upper facial skeleton, the operating surgeon should separate the superfical and deep layers of the deep temporal fascia, reflecting the superficial deep layer and the temporal parietal fascia with the frontal nerve enclosed.

The superfical layer of deep temporal fascia inserts into the lateral border of the zygomatic arch, while deep layer inserts into the medial border of the arch. To elevate and reduce a malar complex fracture through a Gilles approach, posterior to the temporal hairline, the surgeon must pass the elevator deep to the deep layer of the deep temporal fascia and follow the muscle toward its insertion on the coronoid. In this way the elevator is certain to be posterior to the zygomatic arch and malar complex. The temporalis muscle elevates and retracts the mandible (Fig. 3.20).

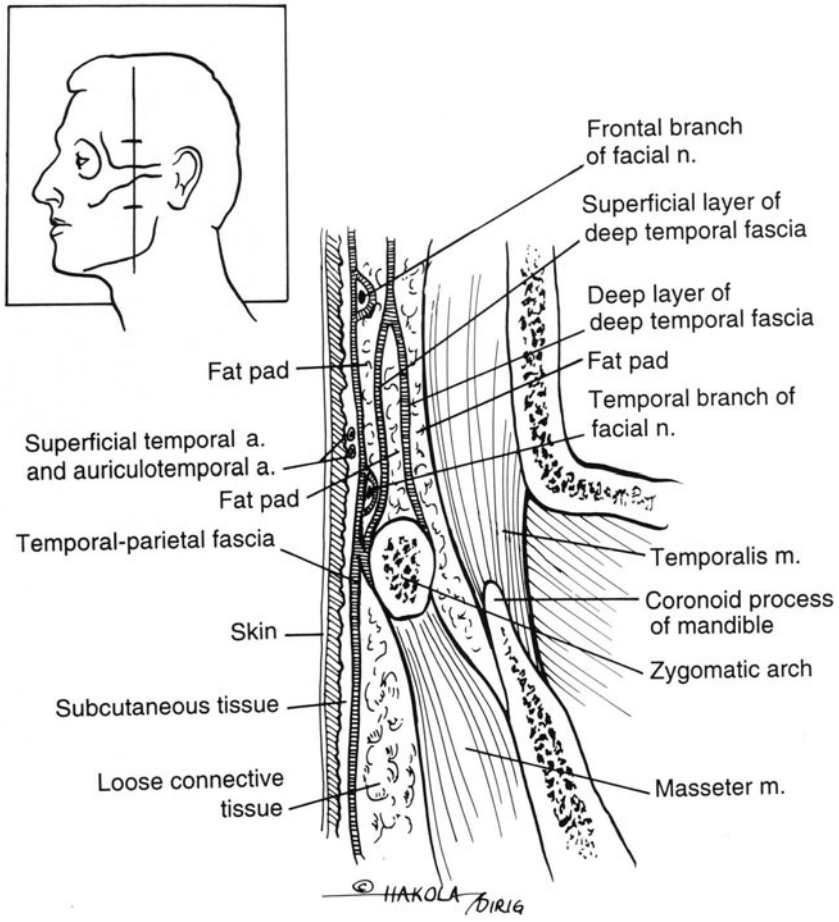


Figure 3.20. Schematic cross-section showing the temporal fascia.

Though there are two pterygoid plates, a medial and a lateral, the pterygoid muscles both take their origin from the lateral pterygoid plate. The lateral (external) pterygoid arises from two heads. The superior head originates from the infratemporal surface of the greater wing of the sphenoid and sweeps downward to its insertion into the articular surface and disk of the temporamandibular joint. The inferior head arises from the lateral surface of the lateral pterygoid plate and runs slightly upward and straight back to insert into the pterygoid fovea on the neck of the mandible. The pull of the muscle exerted on the mandible is primarily forward and medial. The lateral pterygoid is primarily a protruder of the mandible. In fractures or resections of the mandible, this segment is pulled upward and lingually. Understanding this action is essential for the intraoperative alignment of occlusion (Fig. 3.21).

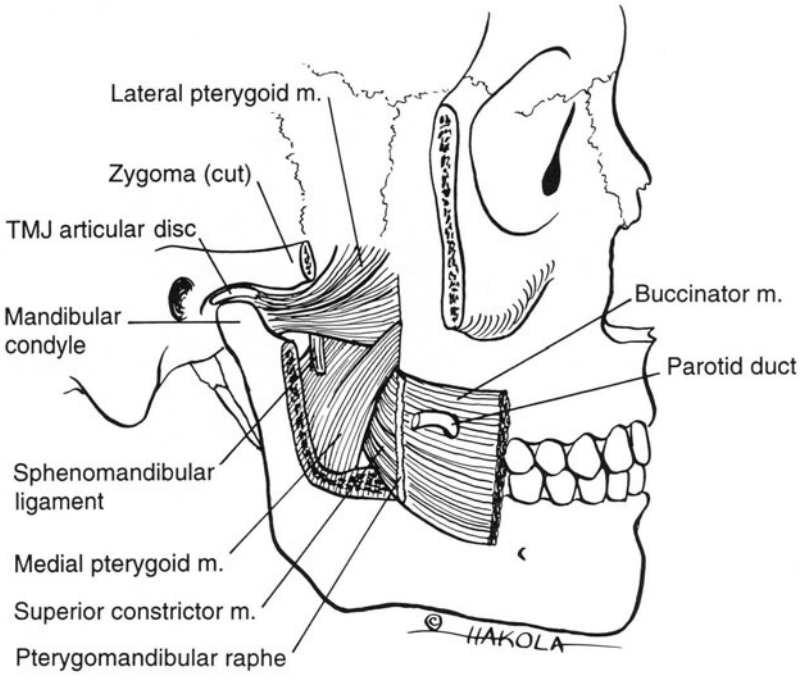


Figure 3.21. The medial and lateral pterygoid muscles.

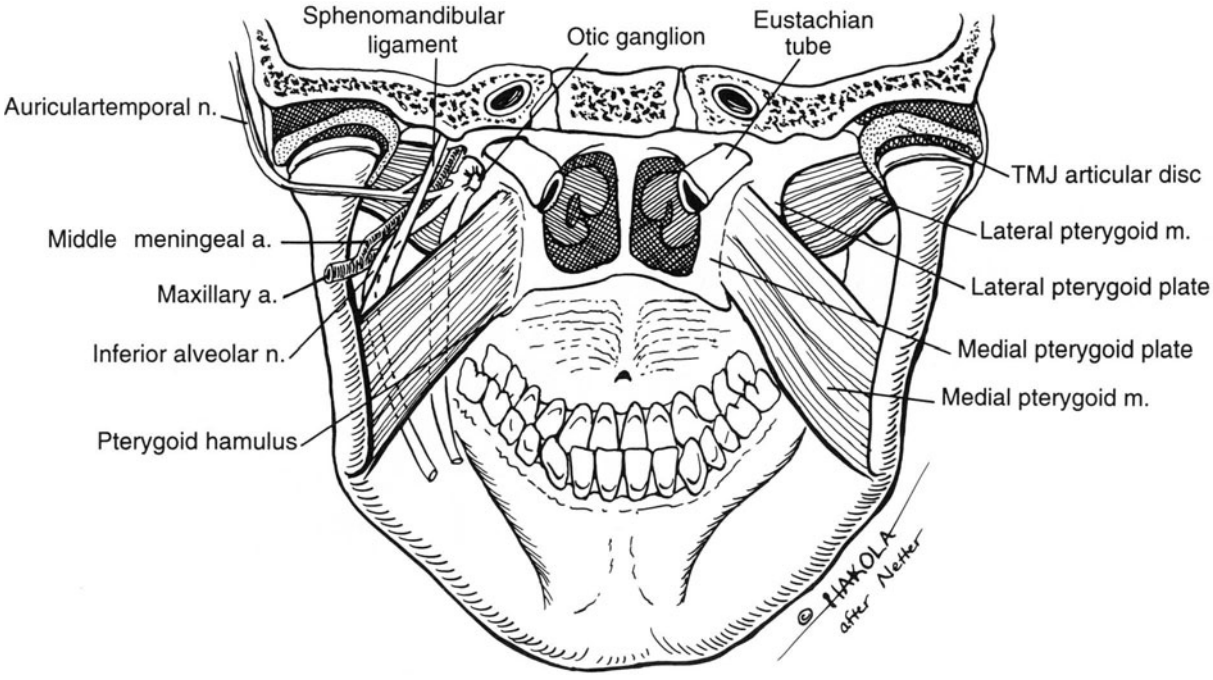


Figure 3.22. Posterior and slightly inferior view of pterygoid muscles and their anatomic relations.

In a LeFort type fracture, the action of the lateral pterygoid pulls the maxillary segment downward and back, resulting in premature contact of the posterior molars. This molar blocking produces an anterior apergnathia (open bite deformity), a frequent presenting sign of the maxillofacial trauma patient.

The medial (internal) pterygoid muscle has its origins from the medial surface of the lateral pterygoid plate (Fig. 3.22). The fibers of this muscle run downward laterally and posteriorly, inserting into the medial and inferior border of the ramus of the mandible, forming a sling with the masseter muscle around the mandibular ramus. The pull of the medial pterygoid is inward and upward, elevating and medially directing the mandible. This pull results in an upward and medial pull of proximal mandibular fracture fragments (Fig. 3.23).

Other Muscles of the Mandibular Region

The geniohyoid muscles are paired muscles whose origin is the mental spine of the inner anterior mandible. The insertion of the muscle is the body of the hyoid bone. The muscle is innervated by the hypoglossal nerve (C.N. XII) as well as fibers from Cervical Nerve 1 (C-1). The effect of this muscle on the mandible is that of a depressor and retractor (Fig. 3.24).

The digastric muscle is composed of a posterior and an anterior belly connected by a long tendon. The posterior belly has its origin on the medial mastoid process. The muscle passes forward and inferiorly, becoming tendonous as it passes through a fascial sling at the level of the hyoid bone. The tendon then courses abruptly upward and forward becoming the anterior belly of the digastric and inserting into the digastric fossa on the lower medial surface of the mandible. The digastric muscle has two innervations. The posterior muscle is innervated by the facial nerve (C.N. VII). The anterior belly is innervated by the trigeminal nerve (C.N. V), by way of the mandibular segment of the nerve, whose inferior alveolar branch gives off the mylehyoid nerve, which further branches to supply the digastrics. The action of the digastric muscle on the mandible is that of a depressor and retractor (Fig. 3.25).

The mylohyoid muscles form a muscular diaphragm, extending from one inner surface of the mandible to the other and posteriorly to the body of the hyoid bone. Above this diaphragm lies the geniohyoid and the muscles of the tongue. Below it lies the digastric muscle and the submandibular glands. The muscle is supplied by the mylohyoid branch of the mandibular nerve. Though the mylohyoid function is that of a tongue elevator during swallowing, its influence upon the mandible is exerted upon the fractured mandible by a resultant lingual inversion of the fractured segment. It is for this reason that placement of a simple inferior border

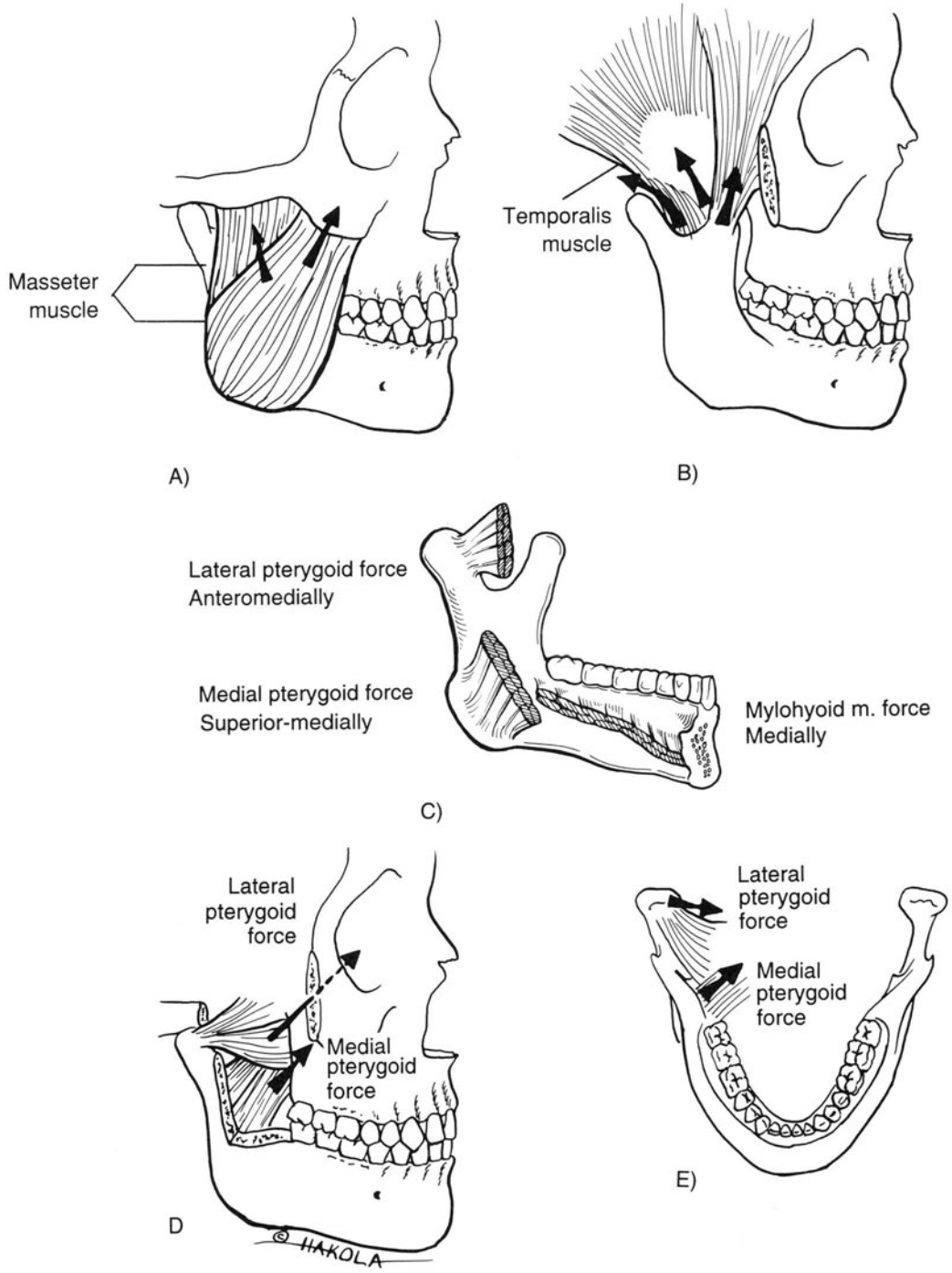


Figure 3.23. Exertion of muscular forces on the mandible. (A) Masseteric muscle force. (B) Temporalis muscle forces. (C) Lingual surface of mandible. (D) and (E) Lateral and medial pterygoid forces.

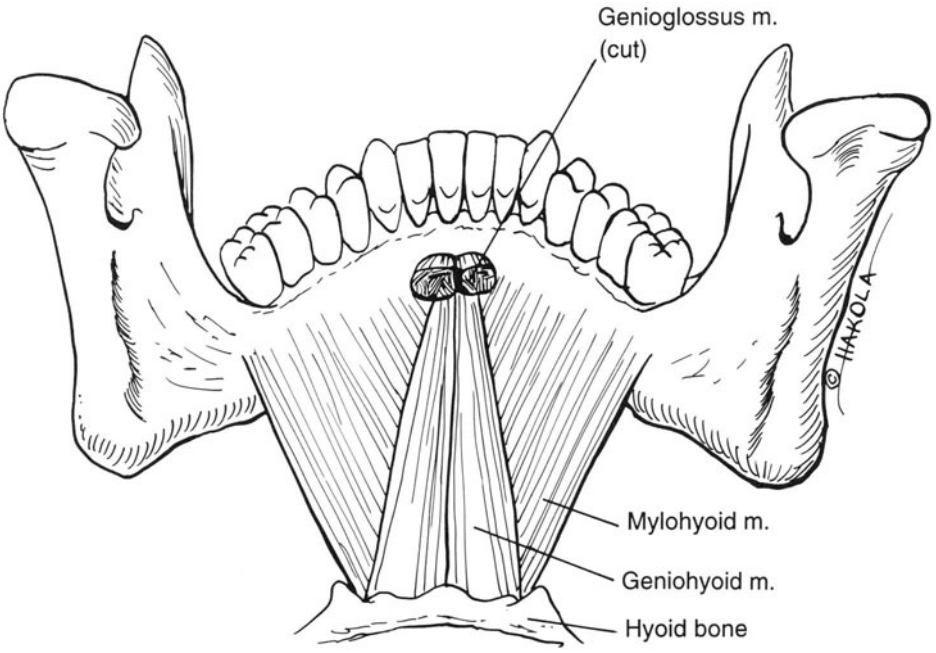


Figure 3.24. Posterior view showing the muscles of the floor of the mouth.

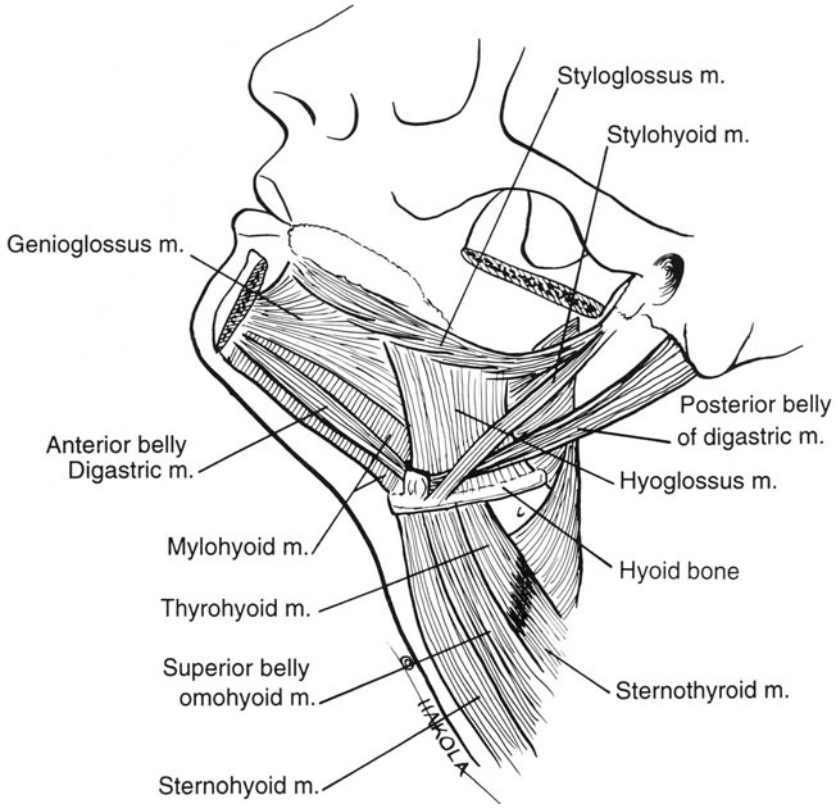


Figure 3.25. The suprahyoid and infrahyoid muscles.

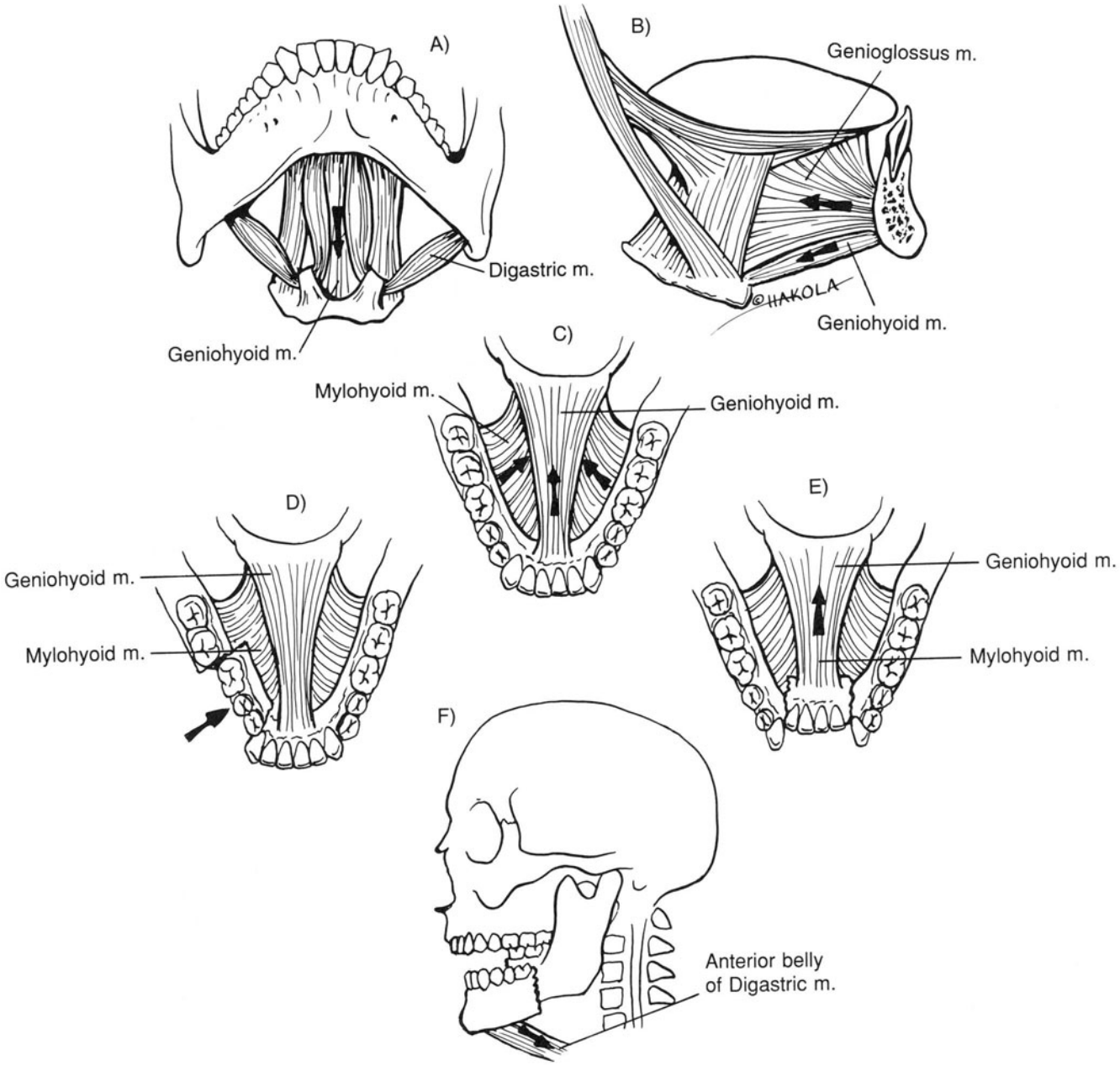


Figure 3.26. Actions of the suprahyoid muscles upon the mandible. Arrows indicate the directional pull of the muscles and displacement of mandibular fracture fragments.

wire on the mandible is insufficient alone to maintain the proper occlusion in a fractured body segment. The resultant actions of the geniohyoid and digastric muscles is to pull anterior fractured segments downward and backward (Fig. 3.26). Overcoming these forces is required to properly reduce bilateral anterior or parasymphaseal fractures. The degree of displacement of mandibular fractures is dependent upon the pull of the musculature with regard to the line of fracture. Those fractures that run vertically from posterior anteriorly and medially are considered to be unfavorable fractures, as the overall muscle effect is to distract the fractured segments. Those fractures that run vertically from the lateral surface inward and posterior have less chance of displacement, as the fragments are being pulled together by the action of the muscles. These fractures are commonly regarded as more favorable (Fig. 3.27).

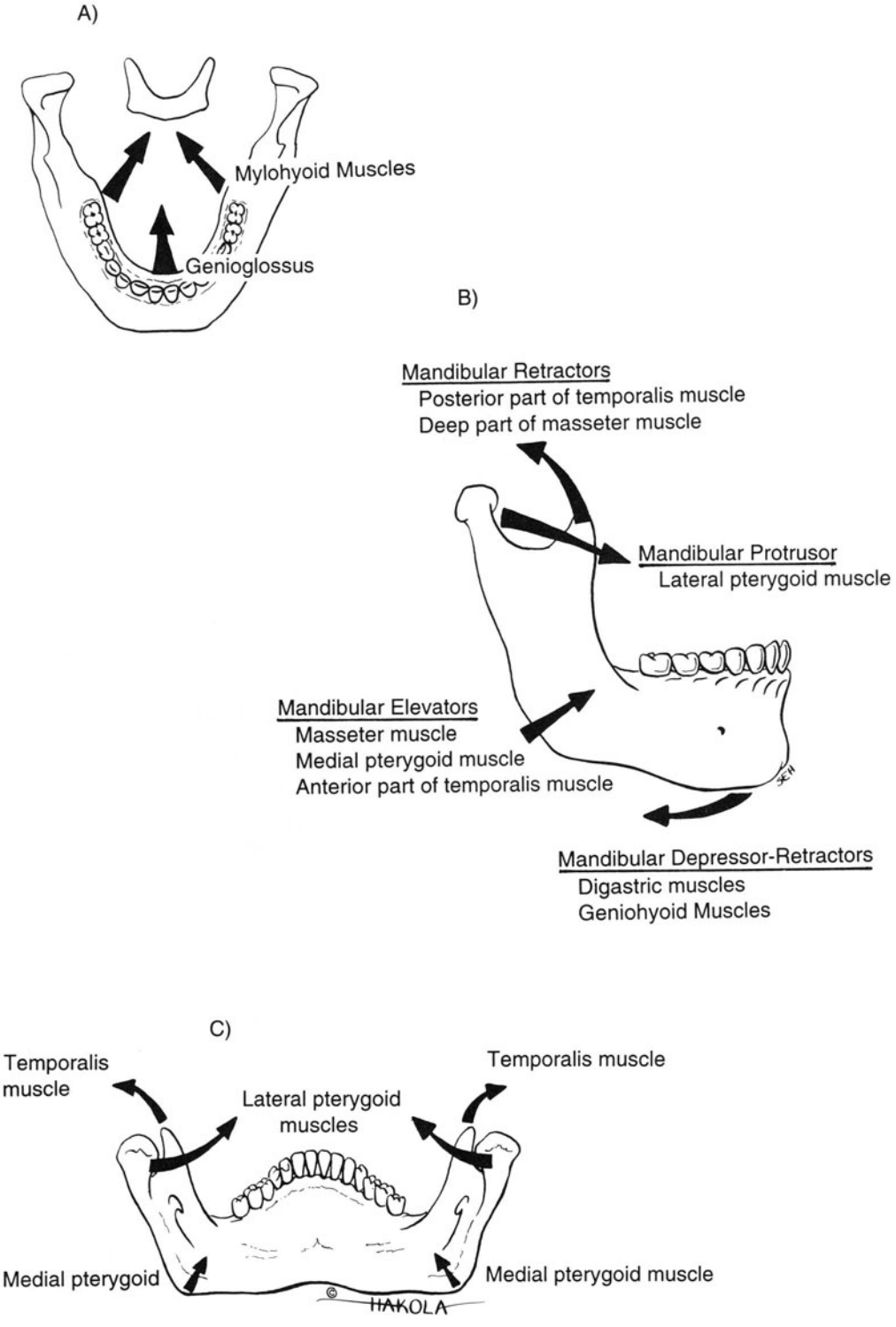


Figure 3.27. Representation of the forces of mandibular musculature upon the mandible. (A) Anterior muscle group. (B) Posterior muscle group and mandibular depressor-retractors. (C) Posterior muscle group.

The Submandibular Region

One can think of the submandibular region as being anterior to the border of the sternocleidomastoid muscle, inferior to the floor of the mouth and superior to the line of the hyoid. A surgical approach to this area would involve an incision approximately one finger breadth below the border of the mandible, extending from the ramus forward to the chin. Directly below the border of the platysma, which overlies the mandible, the facial vein and artery are encountered about halfway between the ramus of the mandible and the chin. The vessels can be palpated at the border of the mandible. Running in the subplatysmal plane is the ramus mandibularis branch of the facial nerve. The nerve runs along the border of the mandible, sometimes above and sometimes below. The nerve crosses superficial to the facial artery and vein. The use of a nerve stimulator in the surgical approach to this area is highly recommended to avoid damaging this nerve. Running from just above the lateral hyoid anteriorly medially and superiorly is the anterior belly of the digastric muscle. Deep to the digastric muscle lies the mylohyoid, which, as previously described, arises from the length of the mylohyoid line along the medial surface of the mandible from the symphysis anteriorly to the third mandibular molar tooth posteriorly. The mylohyoid's central most posterior fibers insert into the upper surface of the hyoid bone.

The submandibular gland forms a U-shape around the free posterior lateral border of the mylohyoid. Just as the posterior ramus of the mandible delineates the parotid into a deep and a superficial segment, so too does the mylohyoid separate the submandibular gland into a deep and a superficial segment. The deep segment of the gland lies on the hypoglossus muscle, which arises from the length of the greater horn of the hyoid and extends superiorly to insert at the side of the tongue. The hypoglossus pulls the sides of the tongue downward. The hypoglossus is innervated by the hypoglossal nerve (C.N. XII), which runs in the space between the deep portion of the submandibular gland and the hypoglossus. Within this space is also found the submandibular ganglion, from which postganglionic fibers pass to the submandibular and sublingual glands. The submandibular duct (Wharton's) exits from the deep part of the gland, running forward first on the hypoglossus and then on the genioglossus to open through the floor of the mouth into the mouth cavity by a small raised papilla close to midline. The lingual nerve, on its way to supply the tongue, runs first above the deep portion of the submandibular gland, then encircles the duct by first lying lateral to, then below, then medial to the duct (Fig. 3.28).

Anterior to the deep portion of the submandibular gland is the sublingual gland. The sublingual gland lies just lateral to the lingual nerve and submandibular duct as they course along the lateral side of the genioglossus. The anterior portion of the gland lies in contact with the anterior portion of the contralateral gland, directly below the mucous membrane of the anterior floor of the

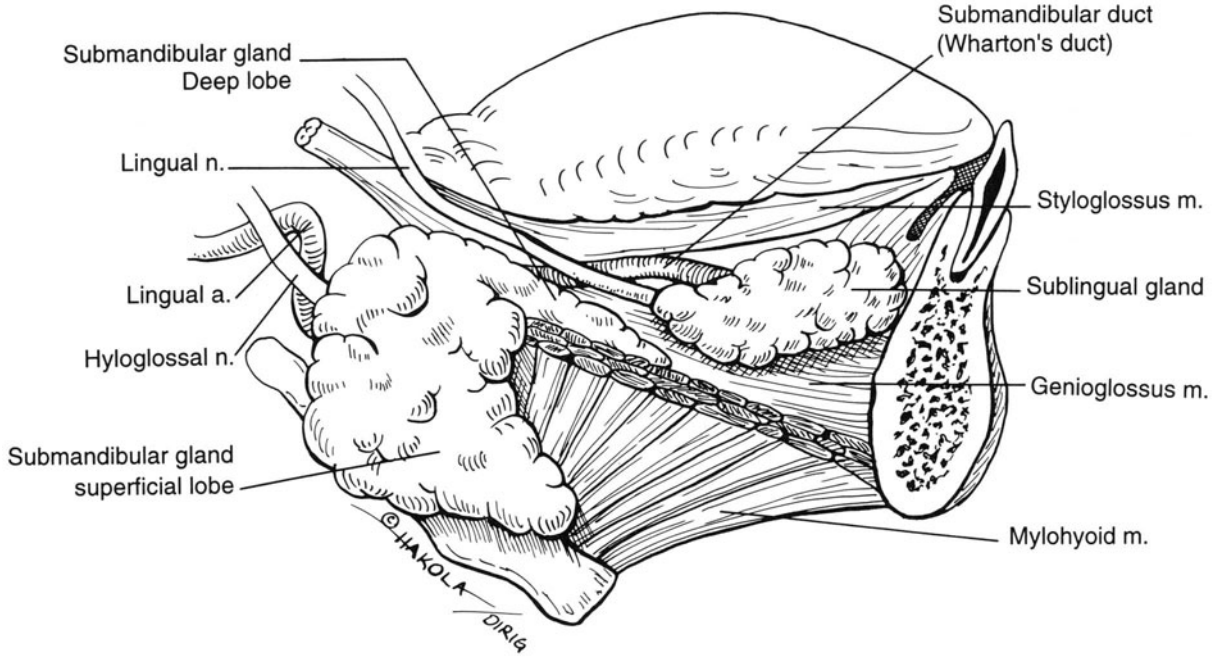


Figure 3.28. Superficial lateral view of the floor of the mouth.

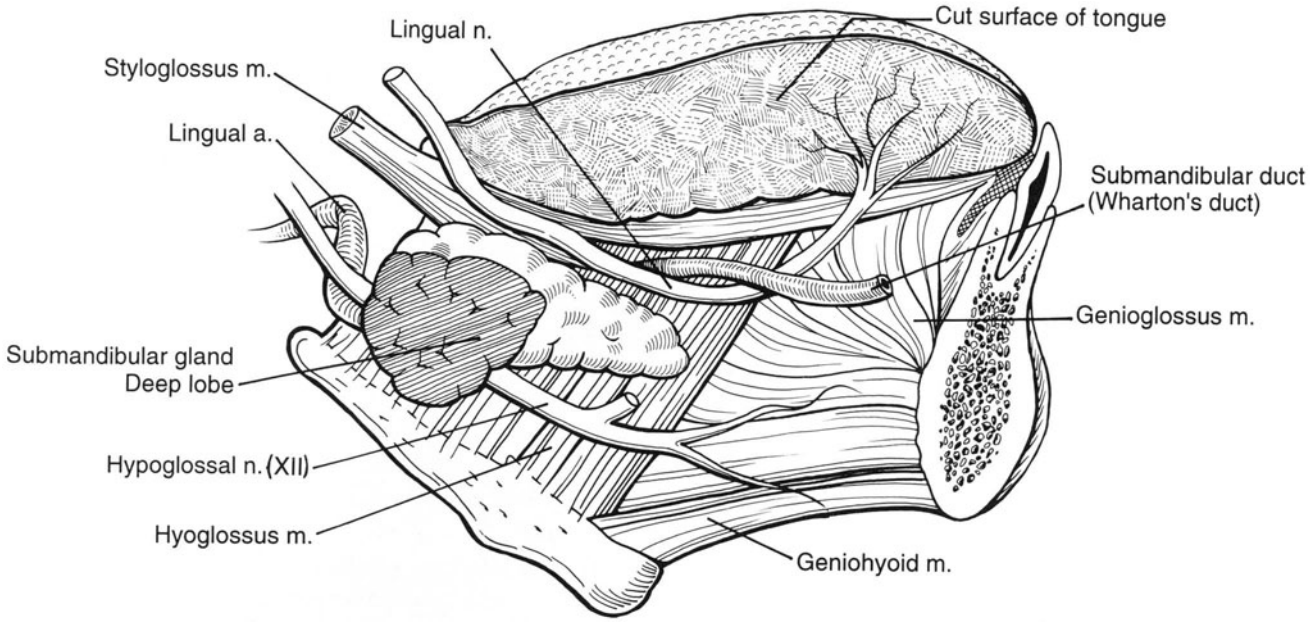


Figure 3.29. Deep lateral view of the floor of the mouth.

mouth. The gland is drained by multiple small ducts, some of which drain into the submandibular duct and others that empty directly into the floor of the mouth. An incision through the mucous membrane just lateral to the tongue passes into the area on the lateral side of the hypoglossus, giving a clear view of the sublingual gland and lingual nerve. The submandibular gland is, however, best operatively approached from below the mandible (Fig. 3.29).

The Tongue

The tongue lies on the floor of the mouth posteriorly attached by its root, through which its nerves and vessels pass. The upper surface of the tongue is covered with mucosa in which reside several different types of papillae. Posteriorly arranged in a "V" at the junction of the anterior two thirds and posterior one third of the tongue lies the large circumvallate papillae bearing numerous taste buds. The anterior two thirds of the tongue is covered with fungiform, conical, and filiform papillae. Of these, only the fungiform papillae are endowed with taste buds. The margins of the tongue are covered with foliate papillae, which are well endowed with taste buds. At the apex to the "V" formed by the circumvallate papillae lies the foramen cecum, a small area indicating the embryologic origin of the thyroid gland (Fig. 3.30).

A mass presenting in this area of the tongue first discovered at puberty may represent a lingual thyroid gland. In some 70–80% of patients this may be the only thyroid gland present.

Posterior to the "V," the pharyngeal surface of the tongue is covered by lymphoid tissue known as the lingual tonsil. Immediately posterior to this at the midline lies the epiglottis, connected to the tongue by a median and two lateral glossoepiglotic folds.

The sensory innervation of the tongue is derived from the first, second, and third branchial arches from which it takes its embryologic origin. The anterior two thirds of the tongue is supplied with general sensation from the lingual nerve branches of the trigeminal nerve. The lingual nerve also carries the taste fibers from the chorda tympani, a branch of the facial nerve. The posterior one third of the tongue is supplied with both taste and general sensation by the glossopharyngeal nerve (C.N. IX). A small area on the lateral posterior lingual tonsil is supplied by the vagus nerve (C.N. X).

The tongue is essentially a muscular organ whose muscles are divided into extrinsic and intrinsic muscle fibers. The extrinsic muscles of the tongue are the genioglossus, the styloglossus, the hypoglossus, and the palatoglossus. The genioglossus muscle extends from the mental spine posteriorly in a fan-shaped manner along the entire area of the tongue from apex to base. The genioglossus acts as a protruder and depressor of the tongue.

The styloglossus originates at the styloid process and stylomandibular ligament. It extends downward and anteriorly to the lateral side of the tongue. The lower fibers of the muscle are related laterally to a portion of the sublingual gland. The styloglossus acts primarily as a retractor of the tongue.

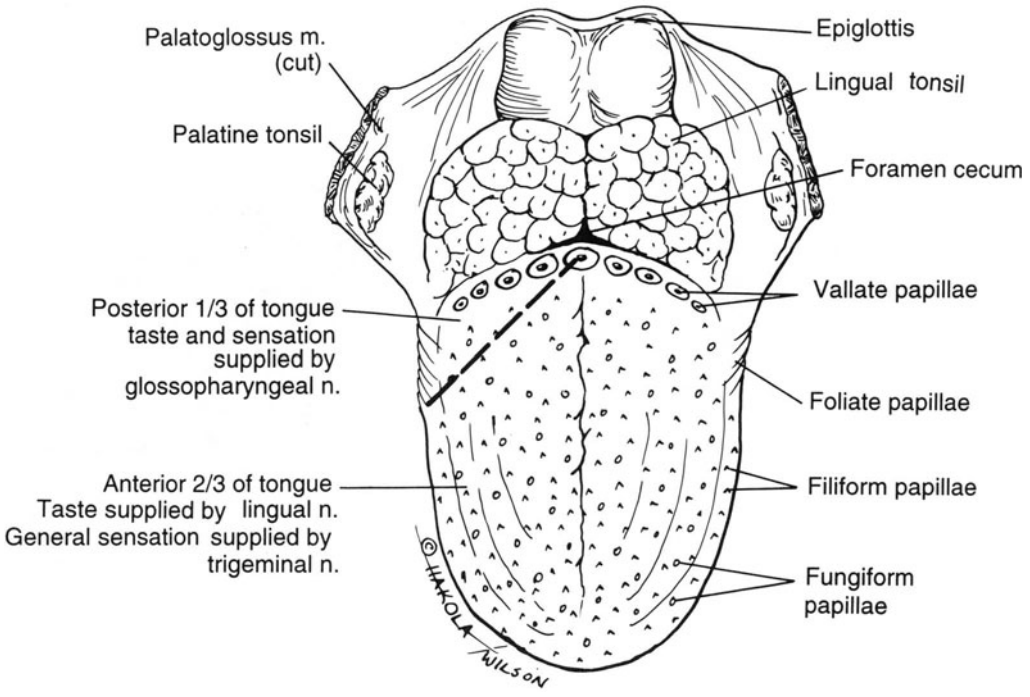


Figure 3.30. Surface anatomy of the tongue, dorsal view. Dashed line represents the junction between the anterior area of sensation and the posterior area of sensation.

The hypoglossus arises from the lateral body and greater cornu of the hyoid. It then passes upward and forward laterally to the posterior genioglossus, where it then interlaces with the styloglossus and intrinsic muscles of the tongue. Its action is that of a tongue depressor.

The palatoglossus (more a muscle of the palate then the tongue) extends from the palatine aponeurosis at the side of the tongue to form the anterior tonsilar pillar. The palatoglossus elevates the root of the tongue. Unlike the other extrinsic and intrinsic muscles of the tongue, which are all supplied by the hypoglossus nerve (C.N. XII), the palatoglossus, being more related to the palatal musculature, is supplied by the vagus nerve (C.N. X).

The intrinsic muscles of the tongue are oriented longitudinally, vertically, and horizontally, with extensive interdigitation of fibers. The intrinsic musculature functions to shape the tongue for swallowing and speech.

The Pharynx

The pharynx may best be considered as a mucosal-covered muscular tube open anteriorly through its entire length until it becomes the closed esophagus at its most inferior aspect. The upper portion of the tube, the nasopharynx, opens to the nasal chambers. The middle portion of the tube, the oropharynx, opens into the mouth. The lower portion of the tube, the laryngeal pharynx, opens to the larynx at the level of the hyoid bone and upper hyoid cartilages. These muscles forming the pharyngeal tube are the superior, middle, and inferior constrictors. Each of these muscles sweeps posteriorly from its anterior origins to join with the corresponding muscle from the opposite side at the pharyngeal raphe of the posterior midline.

The superior constrictor has a broad origin: superiorly from the posterior border of the medial pterygoid plate and its hamulus, inferiorly from the pterygomandibular raphe along the posterior border of the buccinator, and most inferiorly from the posterior part of the hyoid bone over the mandible and the side of the root of the tongue. The connection of the superior constrictor to the buccinator and through the buccinator to the orbicularis oris provides for a continuous ring of musculature from the neck through the lower face. Clefts of the lip break the dynamic balance of this ring and it may be hypothesized that the muscular forces exerted through the broken ring now contribute to the gradual distraction of the cleft segments with every swallow.

The middle constrictor arises from both the lesser and greater cornu of the hyoid bone. Superiorly it overlaps the superior constrictor and is in turn overlapped at its inferior border by the inferior constrictor.

The inferior constrictor arises from the oblique line of the thyroid cartilage, along the tendinous line to the cricoid cartilage. This tendinous line also gives attachment to the thyrohyoid and sternothyroid muscles. The cricopharyngeal portion of the inferior constrictor is also known as the cricopharyngeal muscle (Figs. 3.31 and 3.32).

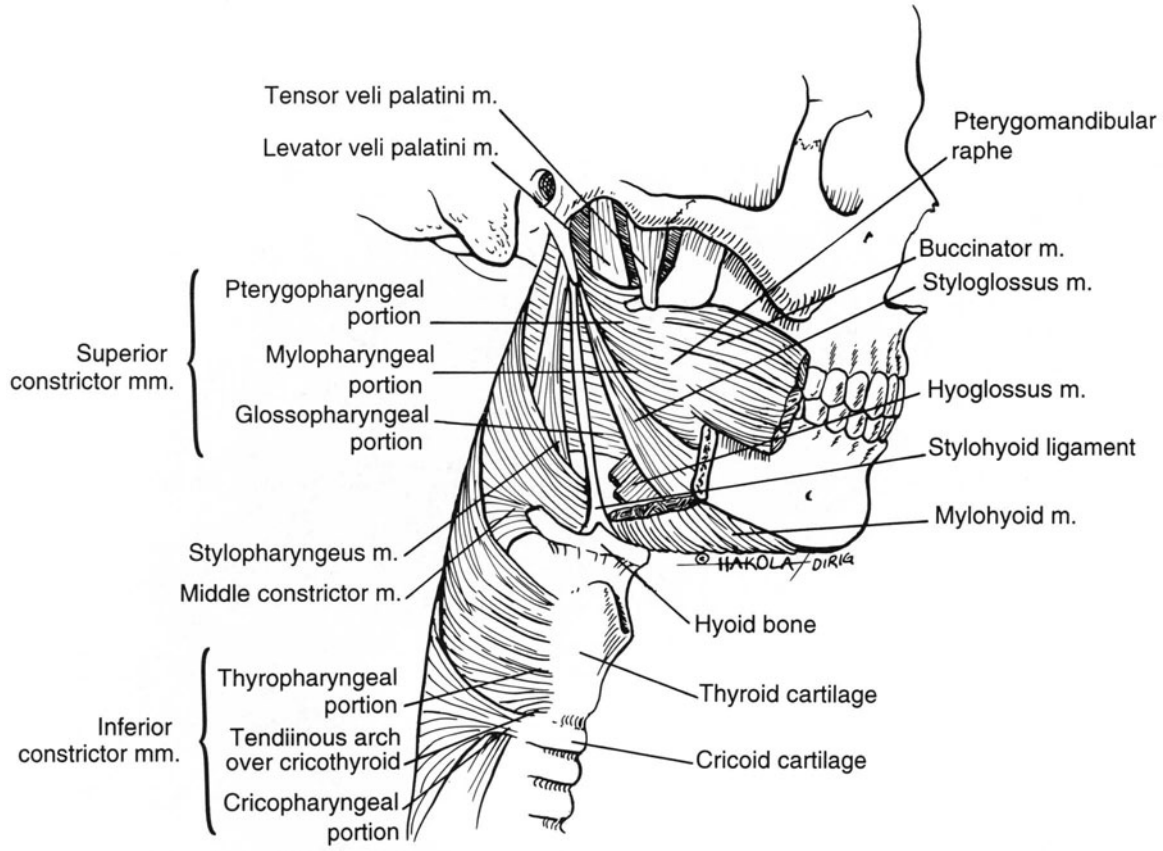


Figure 3.31. View of the pharyngeal constrictors and related muscles.

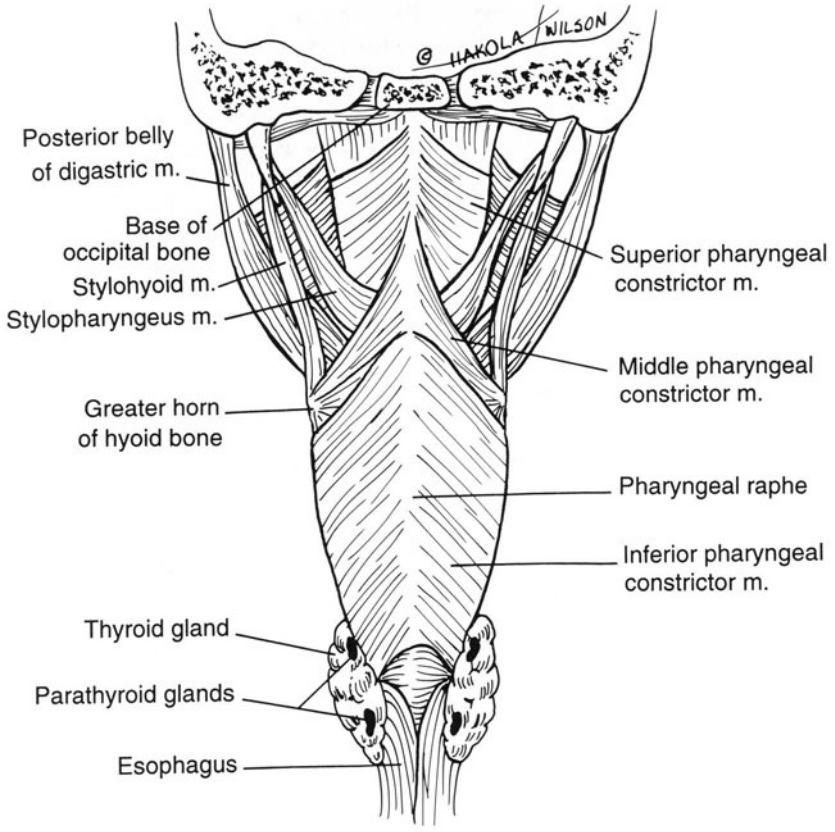


Figure 3.32. Muscles of the pharynx, posterior aspect.

An additional muscle of the pharynx worth mentioning is the stylopharyngeus, which originates from the medial aspect of the styloid process, and passes downward and medially to pass into the pharyngeal wall in the gap between the superior and middle constrictors. The function of the stylopharyngeus muscle is to elevate the pharynx to the esophagus. The innervation of the constrictors, like most of the palatal musculature, is the vagus nerve (C.N. X), while that of the stylopharyngeus is the glossopharyngeal nerve (C.N. IX).

Filling the gap between the upper border of the superior constrictor and the base of the skull are two small muscles, the tensor palati and levator palati. These muscles reinforce the pharyngeal wall in this area, though they are primarily concerned with the soft palate.

The Palate

The palate separates the nasal from the oral pharynx. Proper speech and swallowing functions are directly dependent on the ability of the palate to completely separate these two areas. The palate is composed of a bony hard palate and a muscular soft palate.

The hard palate is formed by the palatine process of the maxilla and the horizontal process of the palatine bones. The maxillary portion is continuous with the alveolar process of the maxilla anteriorly and laterally. The hard palate is divided into a primary portion anterior to the incisive foramen and derived from the premaxilla, and a secondary portion posterior to the incisive foramen derived from the palatine process of each maxilla. The hard palate is covered by mucosa firmly attached to the underlying bone by a tough connective tissue known as the mucoperiosteum. Numerous minor salivary glands are contained in the mucoperiosteum. The blood supply to the hard palate comes anteriorly via the sphenopalatine branch of the maxillary artery, which exits through the incisive canal, and posteriorly via the paired greater palatine branches of the maxillary artery. These branches descend in the greater palatine foramina just medial to the third maxillary molars. More posteriorly there are also the lesser palatine foramen through which emerge minor palatine vessels. The nerve supply to the hard palate comes from the sphenopalatine and greater palatine branches of the maxillary division of the trigeminal nerve (C.N. V). These branches exit with their respective arteries.

The soft palate, or vellum, is composed of a muscular structure bound together by the palatine aponeurosis of connective tissue and covered by mucosa on both its nasal and oral side. The uvula projects downward from the posterior midline of the soft palate. The soft palate musculature is composed of five muscles: the tensor veli palatine, the levator veli palatine, the palatoglossus, the palatopharyngeus, and the muscularis uvulae (Fig. 3.33).

The palatoglossus muscle is the most superficial palatal muscle on the oral side of the soft palate. The palatoglossus fibers, transversely oriented, sweep from the midline of the palate, laterally and inferiorly downward to the lateral margin of the tongue, forming the anterior tonsilar pillar on its way.

The palatopharyngeal muscle is the most superficial of the palatal muscles on the pharyngeal side of the soft

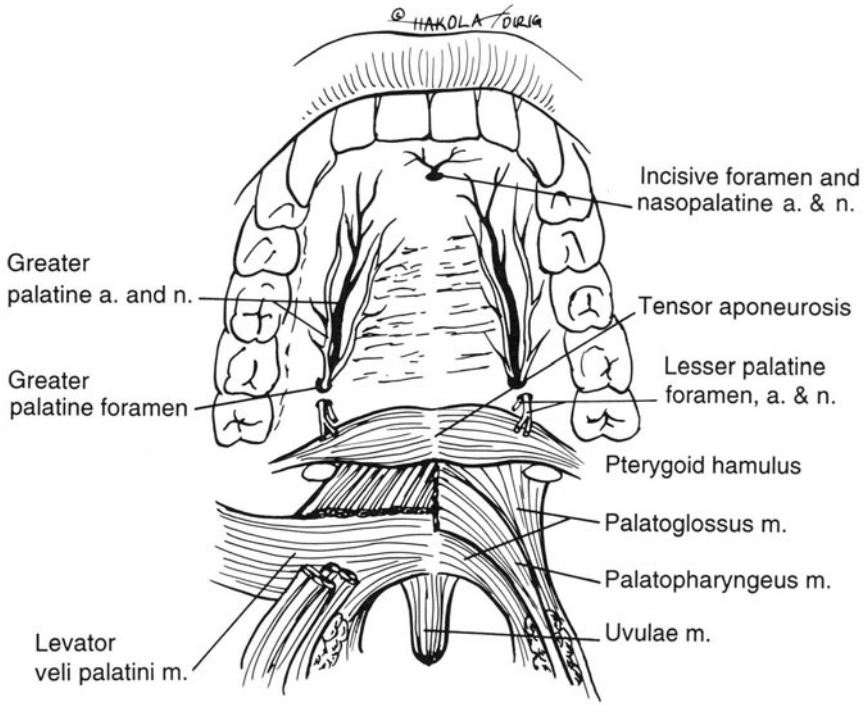


Figure 3.33. Muscular, nervous, and arterial anatomy of the palate.

palate. This muscle, more a pharyngeal muscle then a palatal one, forms the palatal pharyngeal arch along the posterior tonsilar pillar. The muscle sweeps upward into the soft palate, where its anterior and posterior fibers separate to surround the muscularis uvulae. It is the tonsilar portion of this muscle that is elevated and used to fashion a sphincter in a Hines-type pharyngioplasty. The normal action of the palatoglossus and the palatopharyngeus muscle is to draw the soft palate downward and help provide inward lateral pharyngeal wall motion.

The muscularis uvulae is deep to the palatopharyngeus. The fibers of this muscle run longitudinally down the midline of the soft palate and posteriorly into the uvula. The action of the muscularis uvulae is to draw the uvula forward and upward.

The levator veli palatine, the largest muscle of the soft palate, originates at the apex of the petrous bone at the base of the skull and along the medial cartilage of the eustachian tube. It sweeps forward, downward, and medially into the soft palate lying between the muscularis uvulae and the anterior layer of the palatopharyngeus. Constriction of this muscle elevates the palate and pulls open the eustachian tube.

The tensor veli palatini muscle originates broadly from the scaphoid fossa of the medial pterygoid plate and the lateral eustachian tube cartilage. The muscle passes downward between the medial pterygoid muscle and the medial pterygoid plate to the hamulus. The fibers of the muscle are adherent to the hamulus, at which point they continue to sweep medially at a right angle across the palate, attaching themselves to the posterior hard palatal shelf and joining with the contralateral muscle at the soft palate midline. The tensor, as the name implies, tenses the soft palate and depresses it somewhat in the midline. It also serves as the primary opener of the eustachian tube.

In palatal repair, the tightening action of the tensor may inhibit the medial transposition of the palatal muscle. This tension and restriction may be limited by stripping the attachment of the muscle from the hamulus at the time of palatal repair. Unlike the other muscles of the soft palate, which receive their innervation by the vagus nerve (C.N. X), the tensor is innervated by a branch of the mandibular division of the trigeminal nerve (C.N. V).

The Nose

The pyramidal shape of the nose and its gently curving nasal ala is determined by its underlying bony and cartilaginous structure. The upper third of the nose is supported by the paired nasal bones, which are considered separate facial bones. The middle third is supported by the upper lateral cartilages, which extend, tent-like, from their junction with the nasal septum at the dorsum toward the maxilla. The lower third and ala, and the nasal tip, are supported by the lower lateral cartilages.

The nasal septum divides the nasal chamber into right and left nasal cavities (Fig. 3.34). The septum extends

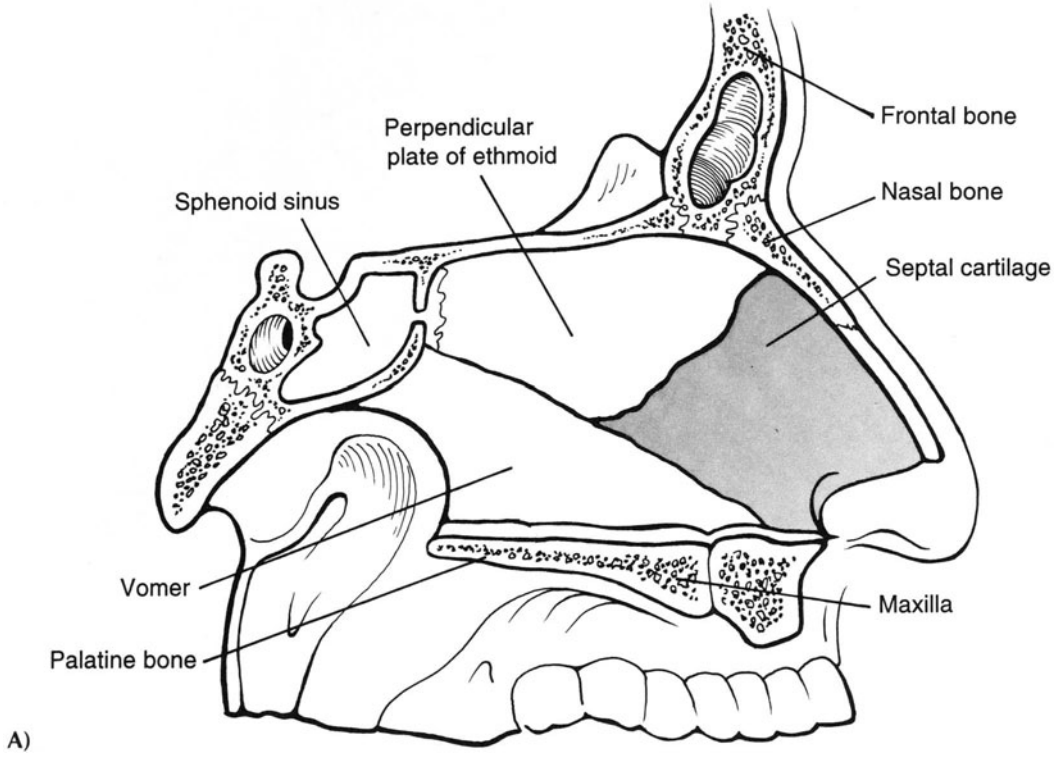


Figure 3.34. (A) The bones and cartilages of the nasal septum.

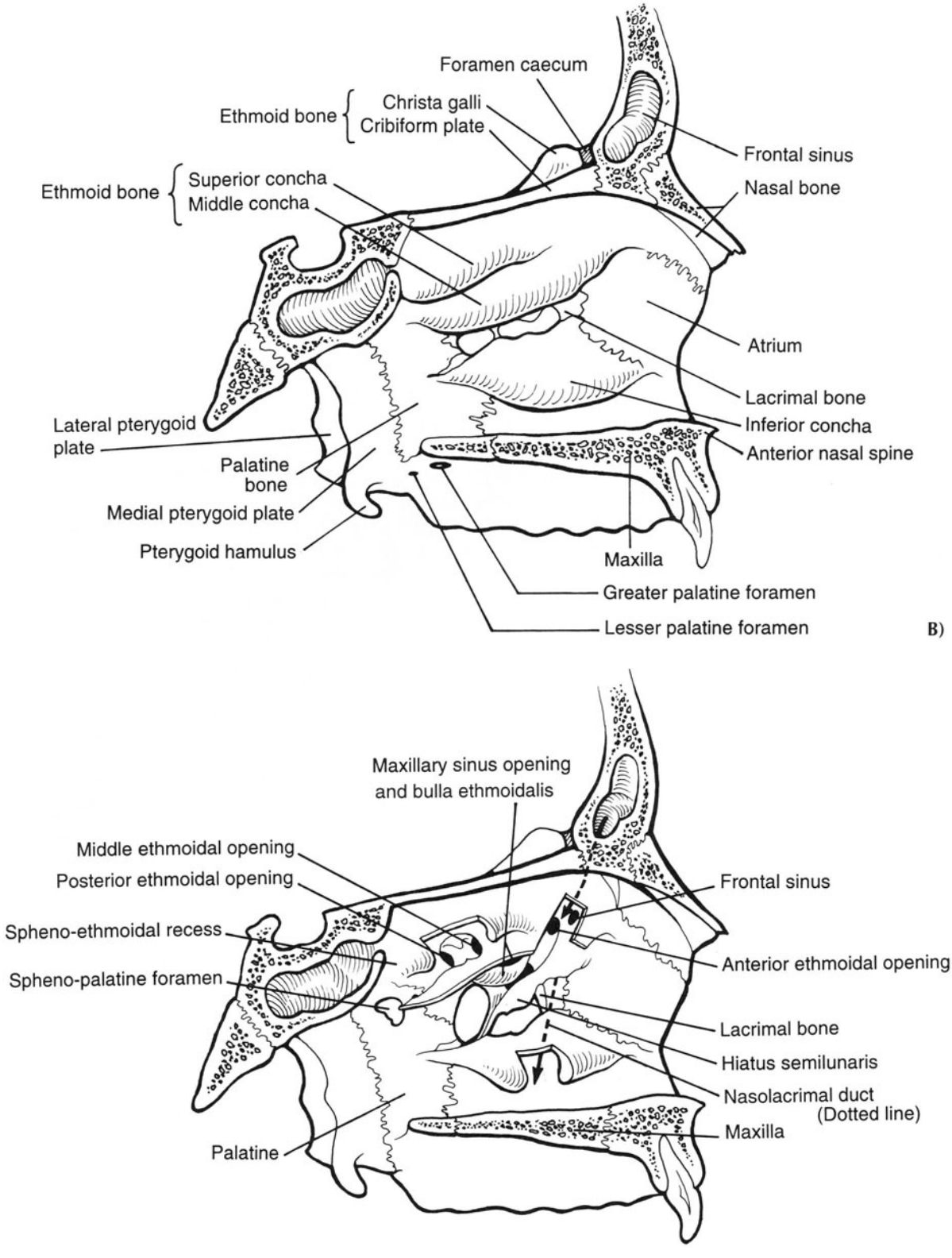


Figure 3.34. *Continued* (B) Bones of the lateral wall of the nasal cavity. (C) Parts of the conchae have been cut away to reveal the openings of the air sinuses. Arrows traverse the frontal air sinus and the nasolacrimal canal.

from the hard palate below, arising from the vomer and perpendicular plate of the ethmoid, to the undersurface of the anterior cranial fossa. A large portion of the septum is formed by the cartilaginous quadrilateral septal cartilage. In early nasal development the nasal cavity may cease its growth while the septum continues to enlarge. Accordingly, the septum constricted in its perpendicular growth will bend and deviate to one side. This deviated septum may obstruct one's airway (Fig. 3.34A).

The roof of the nasal cavity is formed by the frontal bone, the cribriform plate of the ethmoid, and the body of the sphenoid. Special smell receptors, located in the mucous membrane of the upper nasal cavity, transmit their information via rootlets of the olfactory nerve, which pierce the roof of the cribriform plate beneath the crista galli. Sensory innervation of the nose is carried by the sphenopalatine nerve, which innervates the majority of the nasal septum and the anterior third of the hard palate. The anterior ethmoidal nerve supplies sensation to the anterior part of the nasal septum.

The lateral wall of the nose is formed principally by the maxilla and ethmoid. The lateral wall of the nasal cavity separates the nose from the air cells of the paranasal sinuses. Drainage from these sinuses takes place through openings in the lateral nasal wall. The upper part of the lateral nasal wall is formed by the ethmoid, whose air cells form a prominent bulge into the nasal cavity, referred to as the ethmoidal bulla. More anteriorly a thin spur of ethmoid, know as the uncinate process, is present and is separated from the ethmoidal bulla by a shallow groove known as the hiatus semilunaris. The maxillary sinus drains into this groove (Fig. 3.34B and 3.34C).

Overlying the bulla and air cells are delicate scrolls of bone covered by mucus membrane. These scrolls are known as the concha. There are four concha and they are separated from one another by grooves between them, known as meatae. Each meatus is named after the concha to which it is related. From superiorly downward there is the supreme, the superior, middle, and inferior concha. All but the inferior concha are outgrowths of the ethmoidal bone. The inferior concha is considered a separate facial bone. The space between the superior concha in front and the sphenoid behind is the sphenoethmoidal recess.

The sphenoid sinus drains into the sphenoethmoidal recess. The posterior ethmoidal sinus drains into the superior meatus. The anterior and middle ethmoidal sinuses drain through the bulla, while the maxillary antrum and the frontal sinus open into the hiatus semilunaris. The drainage from these areas appears below the middle concha. The nasolacrimal duct opens into the inferior meatus.

The Orbit

The Bony Orbit

The bony orbit is a cross between a pyramid and a cone in shape, with its apex being the most posterior at the entry of the optic nerve through the optic foramen, and its base being the orbital rim. The base, however, does not represent the widest portion of the orbit. The widest portion is found approximately 1.5 cm behind the orbital rim. This corresponds to the widest circumference of the globe found at this point.

The orbit is described as having four walls: a medial and lateral wall, a roof, and a floor. The medial wall of the orbit lies almost directly within the sagittal plane, while the lateral wall diverges laterally outward from apex to base. This results in the orientation of the orbital cone having its apex in the most medial posterior position.

There are eight separate bony portions of the orbit. The roof is composed of the frontal bone and lesser wing of the sphenoid. The lateral wall is composed of the greater wing of the sphenoid and the zygomatic bone. The medial wall is composed of the ethmoidal bone and anteriorly the lacrimal bone, while the floor is made up of the maxilla, comprising the roof of the maxillary sinus and a small posterior segment of the palatine bone. The floor of the orbit, after initially dropping inferiorly, posterior to the inferior rim, starts a gentle upward slope. The continuity of the floor is broken by the inferior orbital fissure, which communicates with the infratemporal space, and the infraorbital canal, which accommodates the infraorbital nerve. The orbital floor is weakest along the canal and accordingly, orbital floor and malar complex fractures frequently occur along this line, resulting in compression of the infraorbital nerve with resultant hypoasthesia. A second area of structural weakness is along the medial orbital wall separating the orbit from the ethmoidal air cells. This area is referred to as the lamina papricia (paper wall) and is frequently blown out in orbital fractures. In addition to the optic foramen, the orbit communicates intracranially via the superior orbital fissure, through which runs the oculomotor, trochlear, and abducens nerves (Fig. 3.35).

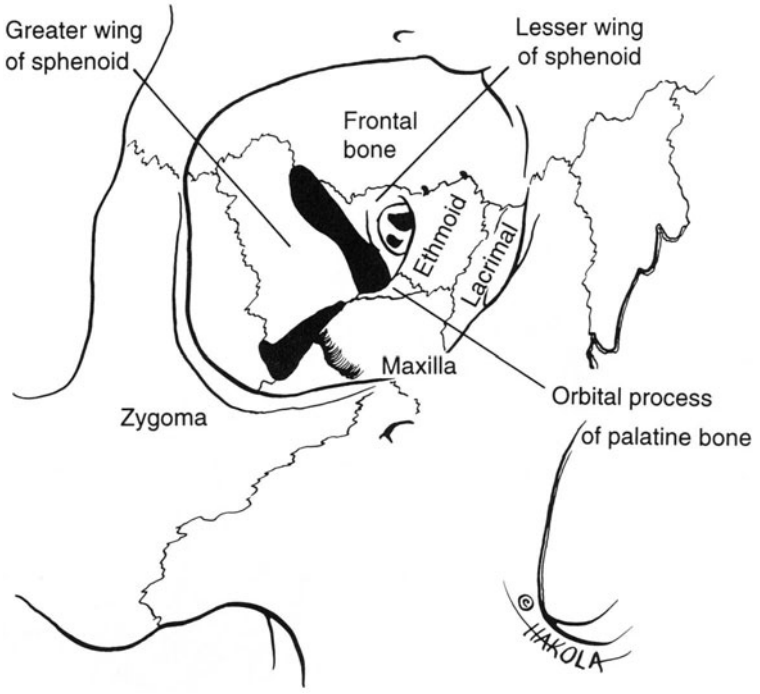


FIGURE 3.35. The boney orbital anatomy.

Operative Landmarks

A lower lid incision is frequently used in maxillofacial surgery to gain access to the bony orbit, most often for repair of orbital blowout fractures. Dissection within the subperiosteal plane elevates the orbital contents from the bony orbit. The optic nerve lies approximately 44 mm posterior to the orbital rim. A helpful landmark to gauge the depth of one's dissection is the posterior wall of the maxillary sinus, which is found at approximately 38 mm from the orbital rim and is easily palpated with an instrument through a floor fracture. The medial wall of the orbit is pierced by the anterior and posterior ethmoidal arteries at approximately 24 mm and 34 mm, respectively, from the orbital rim. Identification of these arteries will provide a more bloodless dissection for the surgeon.

Eyelids

The eyelids consist of several layers. Approached from a skin incision one encounters skin, subcutaneous tissue, orbicularis occuli muscle, orbital septum, tarsus, and conjunctiva. Additionally, behind the orbital septum of the upper lid one encounters first the levator palpebrae superioris muscle, and beneath it, the superior tarsal or Muller's muscle. The levator arises from the bone at the apex of the orbit just above the common tendonous ring and passes forward, lying on the superior rectus to insert into the upper border of the tarsal plate. Muller's muscle is a thin sheet of smooth muscle, sympathetically innervated, which is firmly attached to the posterior surface of the levator and inserts into the upper border of the tarsus. The loss of sympathetic innervation to Muller's muscle, as in Horner's syndrome, leads to a ptosis of the upper lid.

The tarsi are anchored medially and laterally by the medial and lateral canthal ligaments, which connect each plate to the adjacent orbital margin. The medial ligament originates at the medial angles of the two tarsi and extends medially. The majority of the ligamentous fibers pass directly in front of the lacrimal sac to attach to the frontal processes of the maxilla at the anterior lacrimal crest. A few ligamentous fibers will pass behind the sac to attach to the posterior crest. Operative detachment of the canthus from its bony insertion is to be avoided if possible, as the medial angle of the palpebral fissure is difficult to reestablish. A transnasal canthopexy is generally required to reestablish an adequate reattachment.

The lateral canthus originates from the lateral borders of the tarsi and makes its bony attachment to the zygoma at the lateral orbital tubercle.

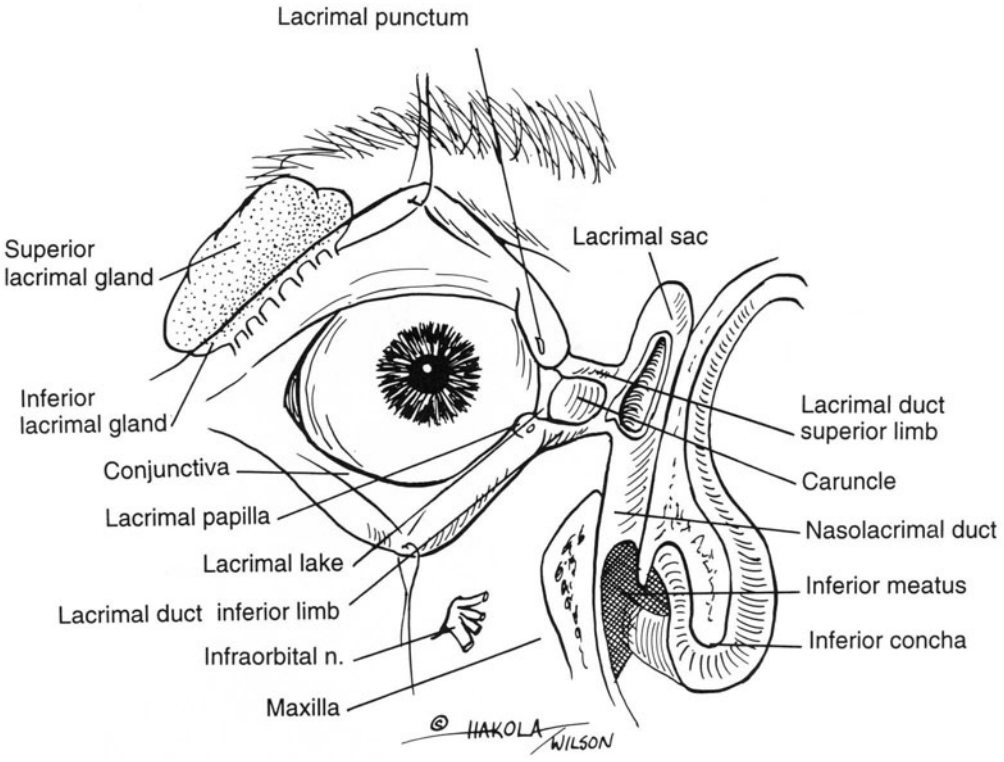


Figure 3.36. The lacrimal apparatus.

The Lacrimal Apparatus

The lacrimal apparatus is composed of the lacrimal gland, the lacrimal ducts, the lacrimal sacs, and the nasolacrimal duct. The lacrimal gland lies in the superior lateral orbit, between the eyeball and the roof of the orbit. The gland is folded about the posterior border of the levator aponeurosis, which separates the gland into an orbital and a palpebral portion. The orbital portion lies directly behind the orbital septum. During an upper blepharoplasty, an inferiorly displaced or "herniated" lacrimal gland may be mistaken for resectable orbital fat. Accordingly, care should be taken to preserve the gland, which can be pexied into its proper position.

Tears produced by the lacrimal gland flow inferiorly and medially across the globe of the eye to accumulate in the lacrimal lake at the medial lower lid. The tears then drain into two small ducts, the lacrimal canaliculi, which lie on the medial end of each eyelid. The visible openings of each of these ducts, the lacrimal punctum, lie on the medial end of each eyelid. Initially, the canaliculi pass vertically away from the lid margins, but after about 2 mm they turn sharply medially to converge on the lacrimal sac. The canaliculi are each approximately 8 mm in length from the punctum to the lacrimal sac. The lacrimal sac, about 12 mm in length, lies in a groove at the anterior end of the medial orbital wall. As described earlier, its upper end is immediately posterior to the medial canthal ligament. From the lacrimal sac, the tears pass through the nasolacrimal duct, entering the nose beneath the inferior meatus (Fig. 3.36).

Operations or disruptions of the lacrimal drainage system via trauma or infection will cause accumulated tears to spill over the lower lid (epiphora).

The lacrimal gland receives its parasympathetic fibers from Cranial Nerve VII, through the sphenopalatine ganglion.

The Periorbita and Rectus Muscles

The boney orbit is lined by a loosely connected periosteum referred to as the periorbita. The periorbita sweeps backward to become continuous with the dural sheath at the optic foramen. Surrounding the area of the optic foramen and superior orbital fissure, the periorbita thickens into a circular structure from which arises the tendonous origins of the four rectus muscles. As the four recti pass anteriorly from the tendonous annular ring to their respective tendonous attachments to the globe, they form a cone of muscle that anatomically defines the retrobulbar space into those structures which are intraconal or extraconal (Fig. 3.37).

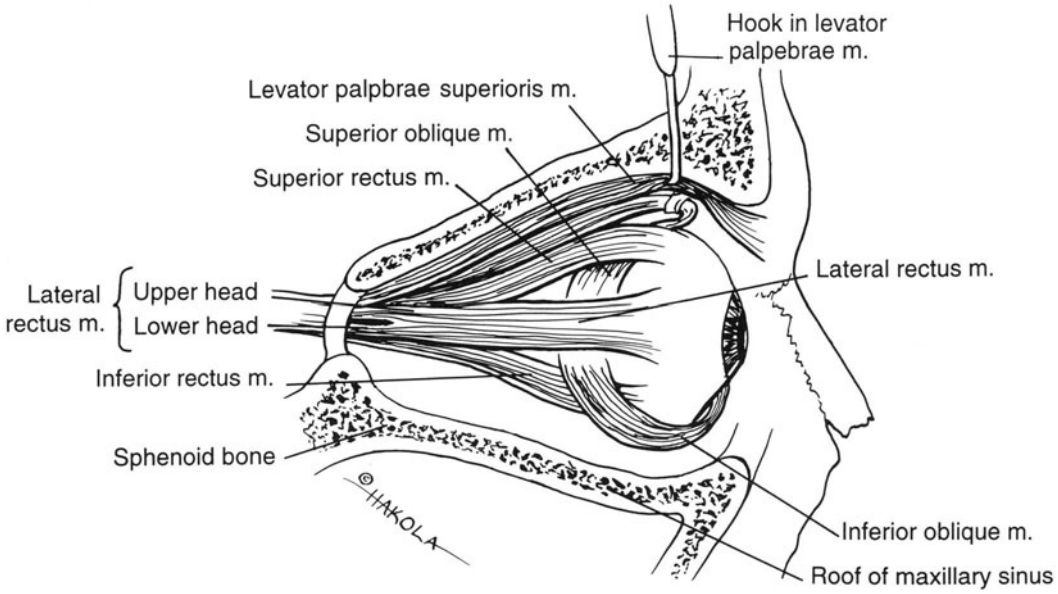


Figure 3.37. Muscles of the right orbit.

The Extraconal Space

Removal of the orbital roof will reveal the superior portion of the extraconal space. Immediately apparent from this approach is the superior orbital fissure. The ophthalmic division of Cranial Nerve V is easily identified within the canal. This sensory nerve divides into three branches (Figs. 3.38, 3.39, and 3.40). The nasociliary branch of the nerve passes into the muscular cone, while the lacrimal and frontal branches continue extraconally.

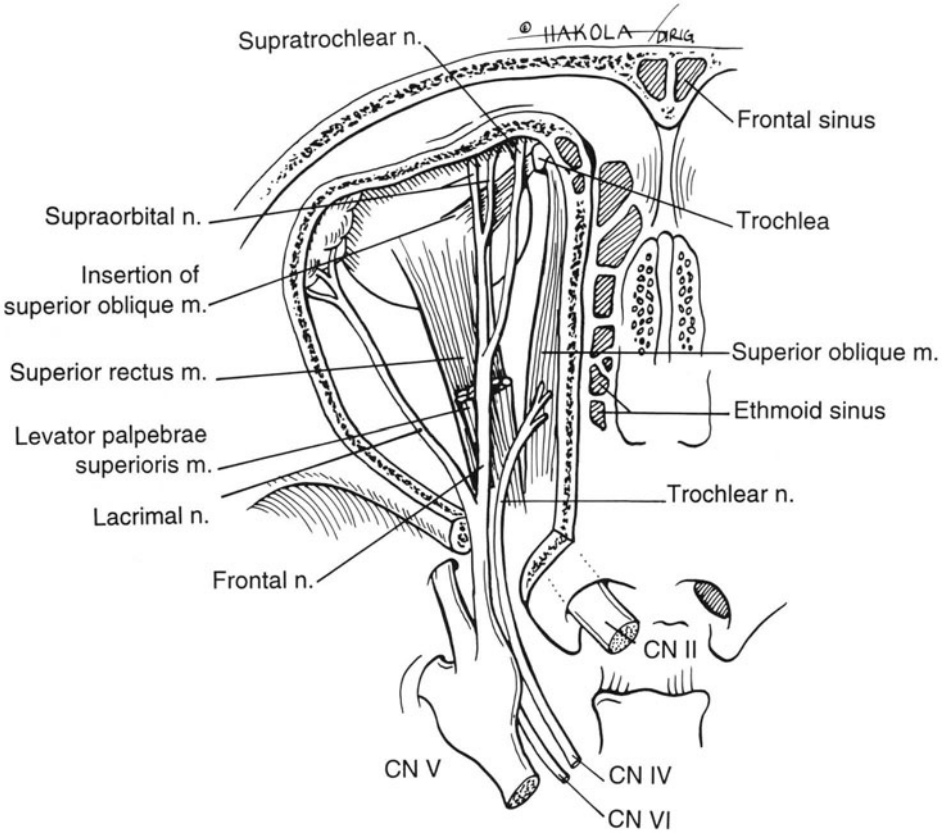


Figure 3.38. Left orbit with roof removed to reveal superior extraconal space.

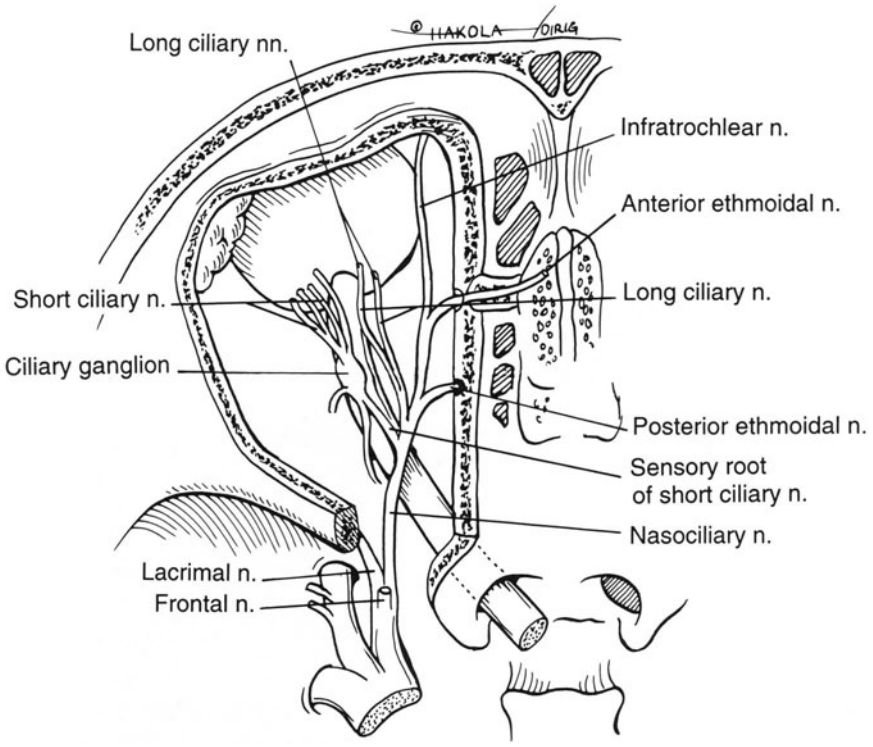


Figure 3.39. Left orbit with roof and extraconal structures removed to reveal intraconal space.

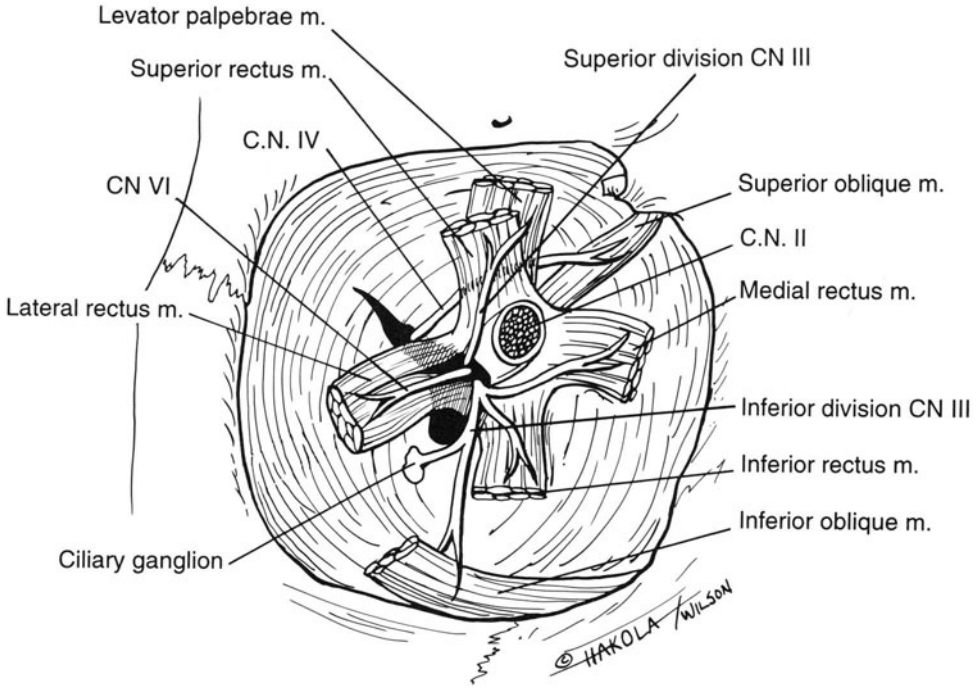


Figure 3.40. Right orbital anterior view of intraconal space.

The lacrimal nerve passes laterally in the orbit to the lacrimal gland and surrounding conjunctiva. The frontal nerve passes anteriorly between the roof of the orbit and the superior levator. Before crossing the superior orbital rim the nerve divides into the supraorbital and supratrochlear nerves, which carry sensation from the skin, the conjunctiva of the medial canthus, the forehead, and the scalp, from as far posterior as the lambdiodal suture. The trochlear nerve (C.N. IV) also passes into the orbit via the superior orbital fissure, but unlike the other nerves to the ocular muscles, the trochlear nerve runs outside the muscular cone, passing medially across the superior levator to innervate the superior oblique muscle. As described earlier, the superior levator runs along the undersurface of the superior orbit into the tarsal plate. Medial and inferior to the superior levator is the superior oblique muscle. The superior oblique muscle arises outside of the annulus, superior and medial to the optic canal. It runs superior and medial to the medial rectus, becoming tendonous prior to passing through the trochlea, a U-shaped cartilage lined by synovial tendon sheath, and attached to the spine of the frontal bone. The superior oblique muscle, on passing through the trochlea, turns sharply posteriorly, laterally, and inferiorly to where it then makes a fan-shaped attachment to the posterior superior quadrant of the globe. The action of this muscle is to pull the pupil into a downward-looking position. When unopposed by the inferior rectus secondary to an oculomotor nerve palsy, the pupil is deflected nasally.

Outside the muscular cone, in the inferior orbit, lies the inferior oblique muscle, which takes its origin just posterior to the medial orbital rim, lateral to the nasal lacrimal canal. The inferior oblique passes laterally, superiorly, and posteriorly, between the periorbita and the inferior rectus muscle, to attach to the posterior lateral aspect of the globe, beneath the lateral rectus muscle. The inferior oblique is innervated by a branch of the oculomotor nerve, which passes through the muscular annulus. The action of the inferior oblique is to rotate the globe into an upward gaze, and when unopposed by the superior oblique, into a lateral gaze as well.

The Intraconal Space

The structures within the intraconal space are best appreciated with regard to their anatomic relationship to the optic nerve. The optic nerve enters the orbit via the optic foramen, approximately 43 mm posterior to the orbital rim. The nerve is accompanied by the ophthalmic artery that runs directly beneath it. The nerve passes through the common tendonous ring to enter the eyeball medial to its posterior pole. The optic nerve is accompanied by an extension of the meninges as far as its entry into the eyeball.

The nasociliary branch of the ophthalmic division of the trigeminal nerve enters the orbit via the optic foramen, passes through the muscular cone lateral to the optic nerve, and then passes superior and medial to the optic nerve, where it then divides into the anterior ethmoidal and infratrochlear nerves. Prior to this division the nerve gives off a sensory root, passing directly into the eyeball. It also branches the two long ciliary nerves, which carry sympathetic fibers from the internal carotid artery directly into the eyeball. Sympathetic stimulation of these nerves functions to dilate the pupil. The anterior ethmoidal nerve carries sensation from the ethmoidal air cells and the tip of the nose. The infratrochlear nerve carries sensation from the bridge and the side of the nose (Fig. 3.39).

The oculomotor nerve (C.N. III) enters the orbit via the superior orbital fissure and immediately passes within the muscular cone. The nerve then divides into the motor branches for all the ocular muscles except the lateral rectus and superior oblique. A convenient formula to remember for the ocular muscle innervation is LR_6SO_4, indicating the lateral rectus muscle innervation by Cranial Nerve VI and the superior oblique by Cranial Nerve IV. The remainder of the orbital muscles are all innervated by Cranial Nerve III (Fig. 3.40).

The ophthalmic artery (Fig. 3.41) enters the orbit through the optic foramen inferior to the optic nerve. The artery passes through the tendonous ring into the muscular cone. The artery first branches into the central artery of the retina and numerous ciliary branches that supply the eyeball. The artery then spirals laterally, sending a branch to the lateral orbital wall. The artery then crosses over the optic nerve to the medial wall of the orbit. There are numerous branches of the artery that supply all the ocular structures, including the conjunctiva and the skin of the lids. The ophthalmic veins run along with the arteries and coalesce to form the superior and inferior ophthalmic veins. The superior ophthalmic vein passes back through the superior orbital fissure and into the cavernous sinus. The inferior ophthalmic vein passes back through the inferior orbital fissure to communicate with the plexus and cavernous sinus. It is this arrangement of venous drainage that makes orbital and upper facial infections potentially so dangerous, as bacteria can travel from an extra-to intracranial location with resultant meningitis (Fig. 3.42).

The Cranial Facial Skeleton

As discussed in the chapter on embryology, the cranial facial skeleton has its origins in both membranous and cartilaginous bone. There are 10 cranial bones. They are as follows (Figs. 3.43 and 3.44):

One frontal
Two ethmoid
Two sphenoid

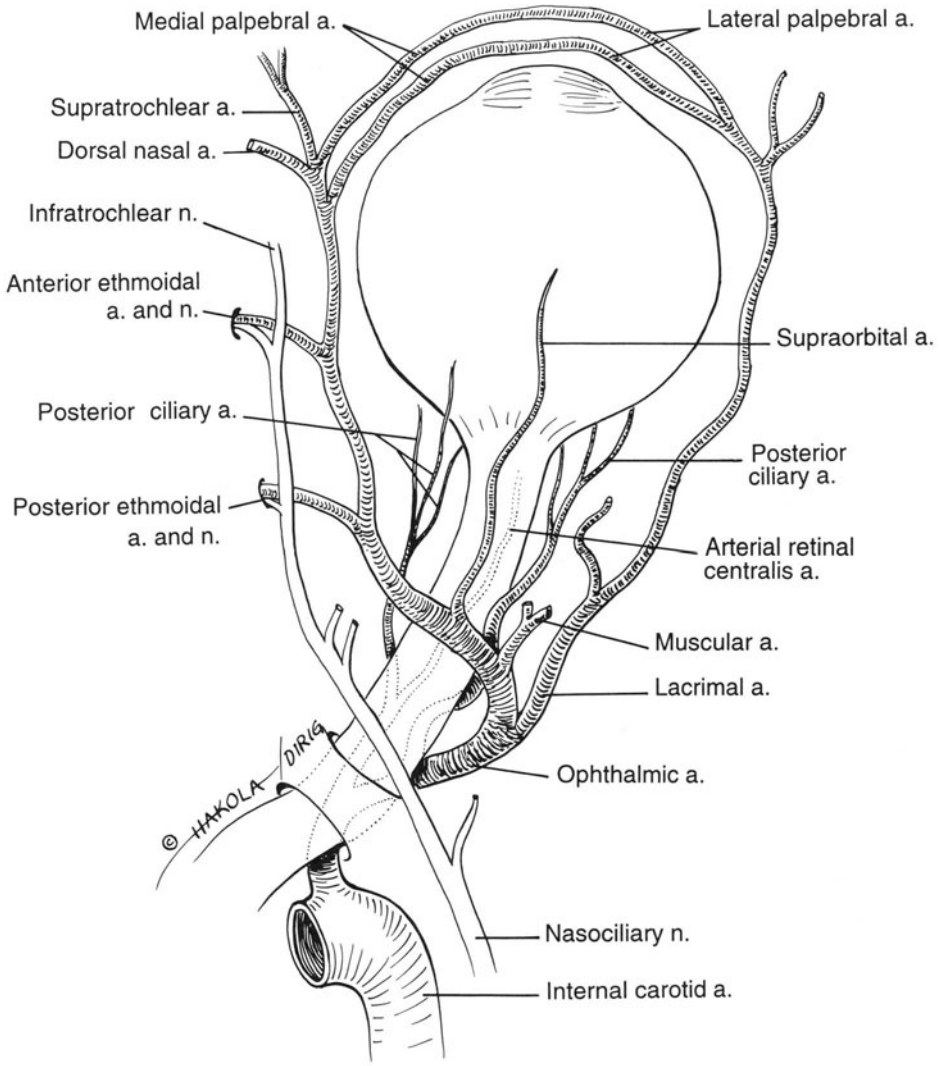


FIGURE 3.41. Arterial circulation of the right orbit.

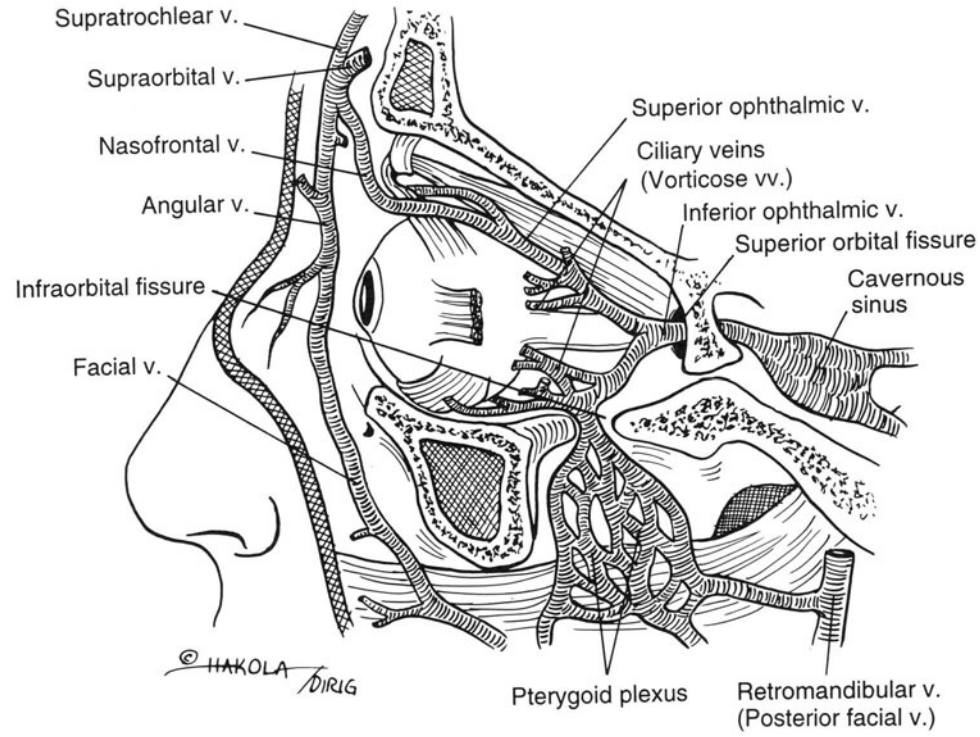


FIGURE 3.42. Venous circulation of the orbit, lateral view. Note the ophthalmic veins communicating with the cavernous sinus through the superior orbital fissure. The inferior ophthalmic vein communicates through the infraorbital fissure with the pterygoid plexus.

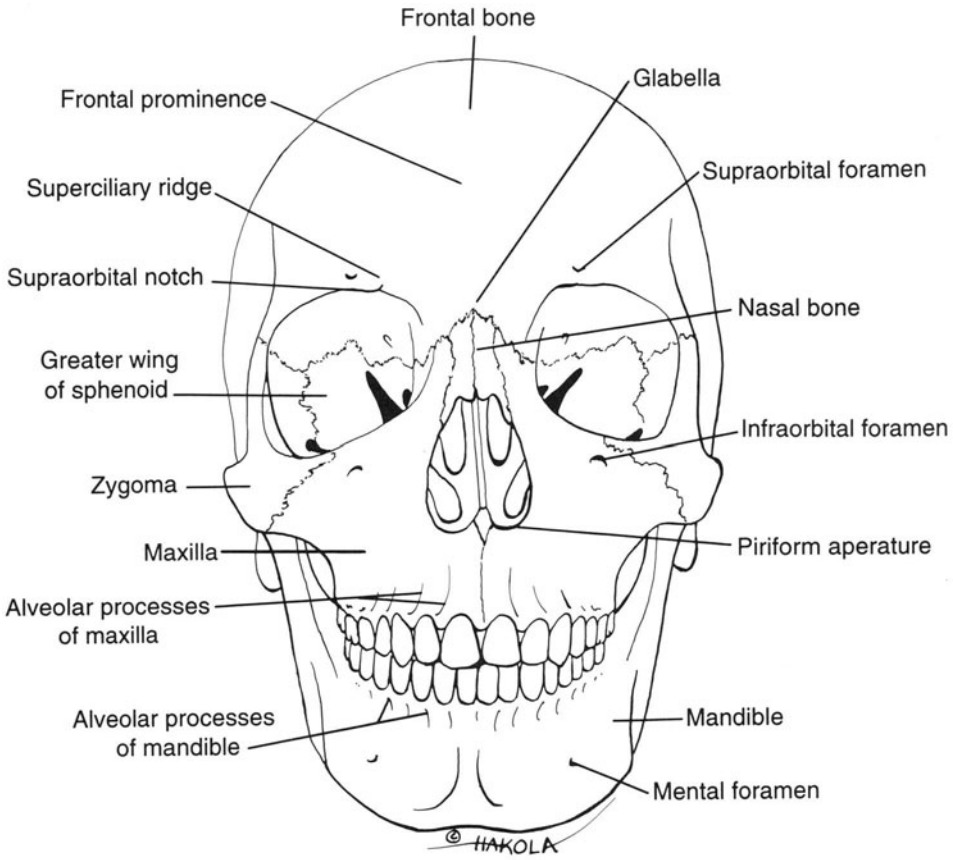


Figure 3.43. The skull, anterior view.

One occipital
Two parietal
Two temporal

The facial bones are 15 in number, and are listed as follows:

Two nasal
Two lacrimal
Two inferior nasal concha
Two maxilla
Two zygomatic
Two palatine
Two mandible
One vomer

It is of interest to note that the superior and middle nasal concha are part of the ethmoid and are hence classified as cranial bones, while the inferior nasal conchae are independent facial bones (Fig. 3.45). The cranial bones are composed of inner and outer cortical tables separated by vascular cancelous bone, making up the diplopic space (Fig. 3.46). As the inner table of cranial bone is thin, care must be taken when harvesting bone grafts not to lever the osteotome against the inner table, as this may lead to a depressed skull fracture. The cranial bones are joined along their respective suture lines. The coronal suture joins the frontal bone to the parietal bones. The lambdoidal suture joins the occiput to the parietal bones. Running anterior to posterior at the midline, the sagittal suture extends between the frontal and lambdoidal suture lines (Fig. 3.47). The point of intersection of the coronal and sagittal sutures is the bregma. The point of intersection between the lambdoidal and sagittal sutures is the lambda. Directly below the sagittal suture runs the sagittal sinus, the major venous drainage of the brain. Cranial bone should not be harvested from the sagittal midline, to avoid disruption of the sagittal sinus. The dome of the cranium extends from the superciliary arches, posteriorly to the external protuberance and superior nuchal line.

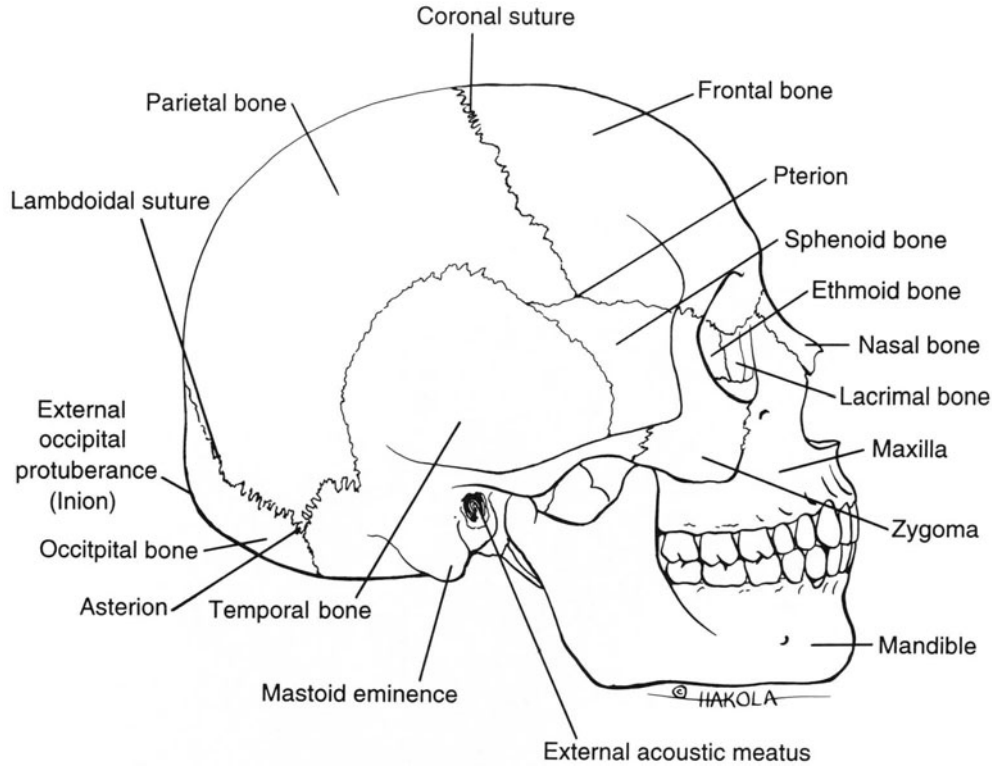


Figure 3.44. Lateral view of the skull.

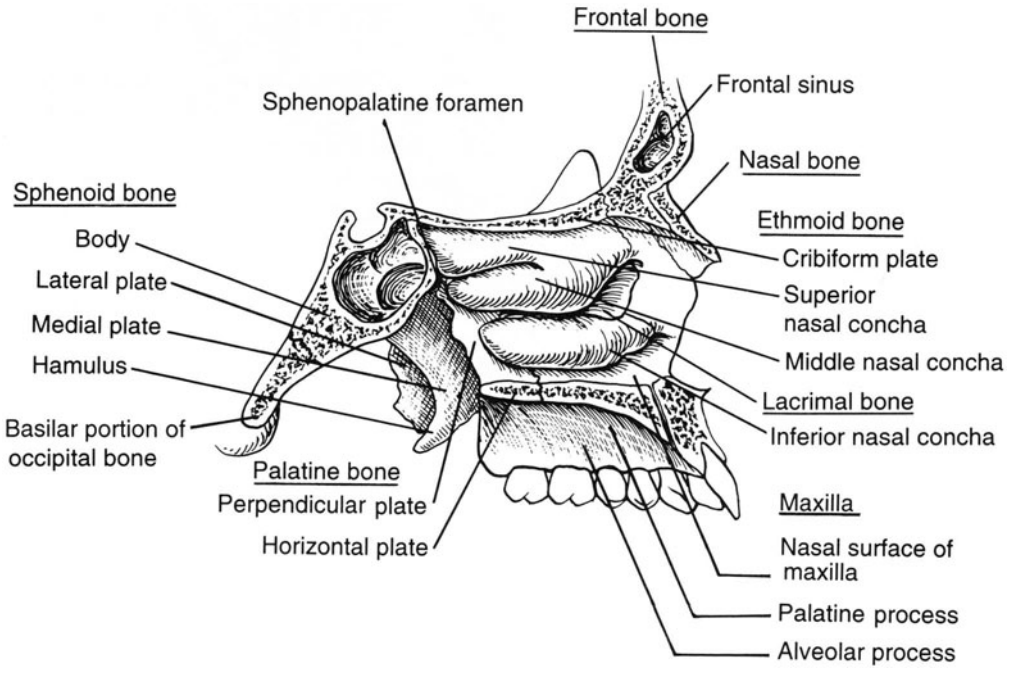


Figure 3.45. Nasal region with vomer and perpendicular plate of ethmoid bone removed.

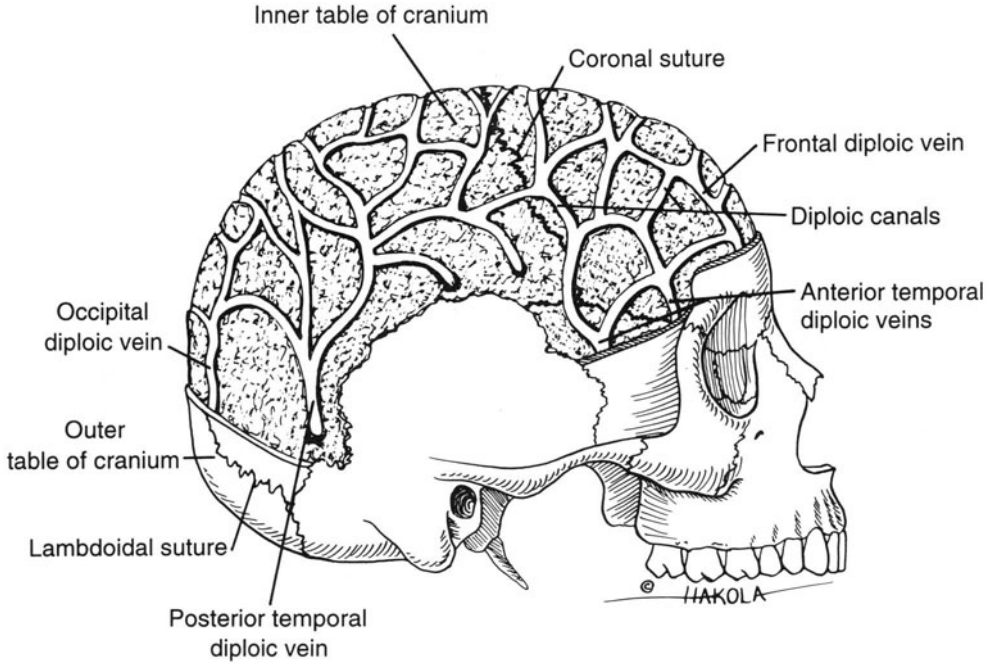


Figure 3.46. The cranial architecture and the four diploic veins.

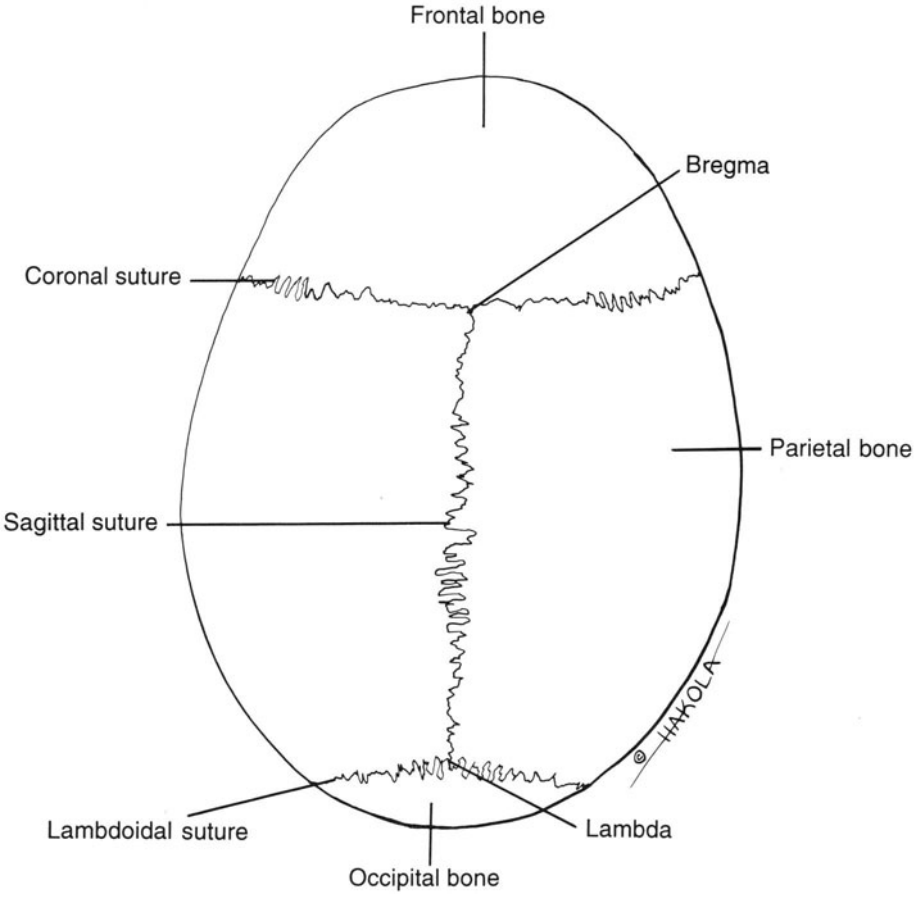


Figure 3.47. Superior aspect of the skull.

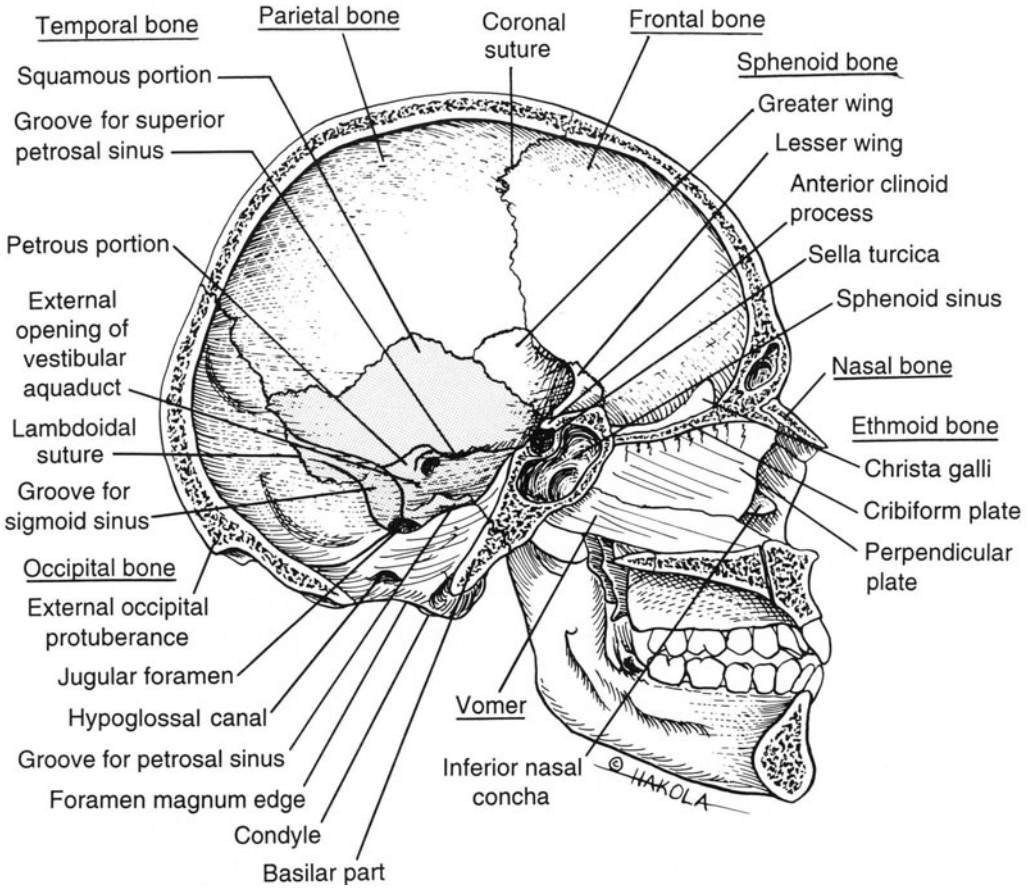


Figure 3.48. Midsagittal section of skull.

The temporal fossa includes portions of the frontal and parietal bones, as well as the squamous portion of the temporal bone, and greater wing of the sphenoid. The temporal bone itself may be divided into a squamous portion and an inferior petrous portion, as well as the mastoid, styloid, and zygomatic processes (Figs. 3.48 and 3.49).

The basal aspect of the skull may be considered from anterior to posterior. The alveolar process of the maxilla is first encountered along with the teeth of the upper jaw. Proceeding posteriorly from the alveolar process one encounters the palatine process of the maxilla, fused at the midline to make up the anterior two thirds of the hard palate. The posterior one third of the hard palate is formed by the horizontal plate of the palatine bone, which lies between the medial pterygoid plates of the sphenoid bone. The lesser palatine nerves pierce the palatine bone on the way to the soft palate. The sphenoid bone fills the cranial base between the occipital bone and the palatine bone. Centrally the paired choanae join with the vomer midline. Extending laterally one encounters the pterygoid process, which includes the medial and lateral pterygoid plates, as well as the hamulus. More laterally, the greater wing of the sphenoid joins with the temporal bone laterally and posteriorly. Piercing the sphenoid bone are the foramen ovale, through which passes the mandibular nerve, and the foramen spinosum, through which passes the middle meningeal artery.

Posteriorly, at midline, the sphenoid joins with the basalar part of the occipital bone, anterior to the foramen magnum. Laterally, the sphenoid is separated from the occipital bone by the petrous portion of the temporal bone. At the junction of the posterior sphenoid and the petrous portion is the foramen lacerum, through which runs the internal carotid artery (Fig. 3.50).

The petrous portion of the temporal bone includes the stylomastoid foramen, through which exits the facial nerve, and the jugular foramen, through which exits the internal jugular vein and Cranial Nerves IX, X, and XI. Posterior to the petrous portion of the temporal bone, the remainder of the skull base is composed of the occipital bone.

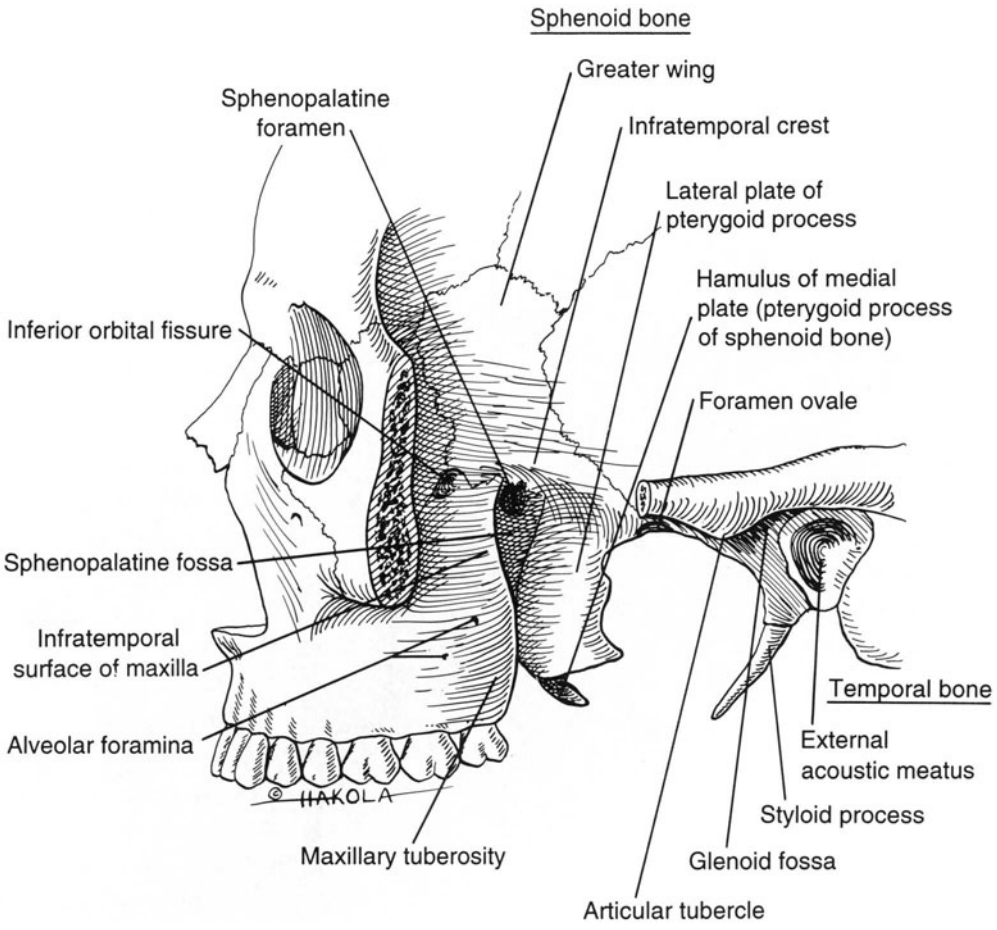


Figure 3.49. Infratemporal fossa exposed with zygomatic arch removed.

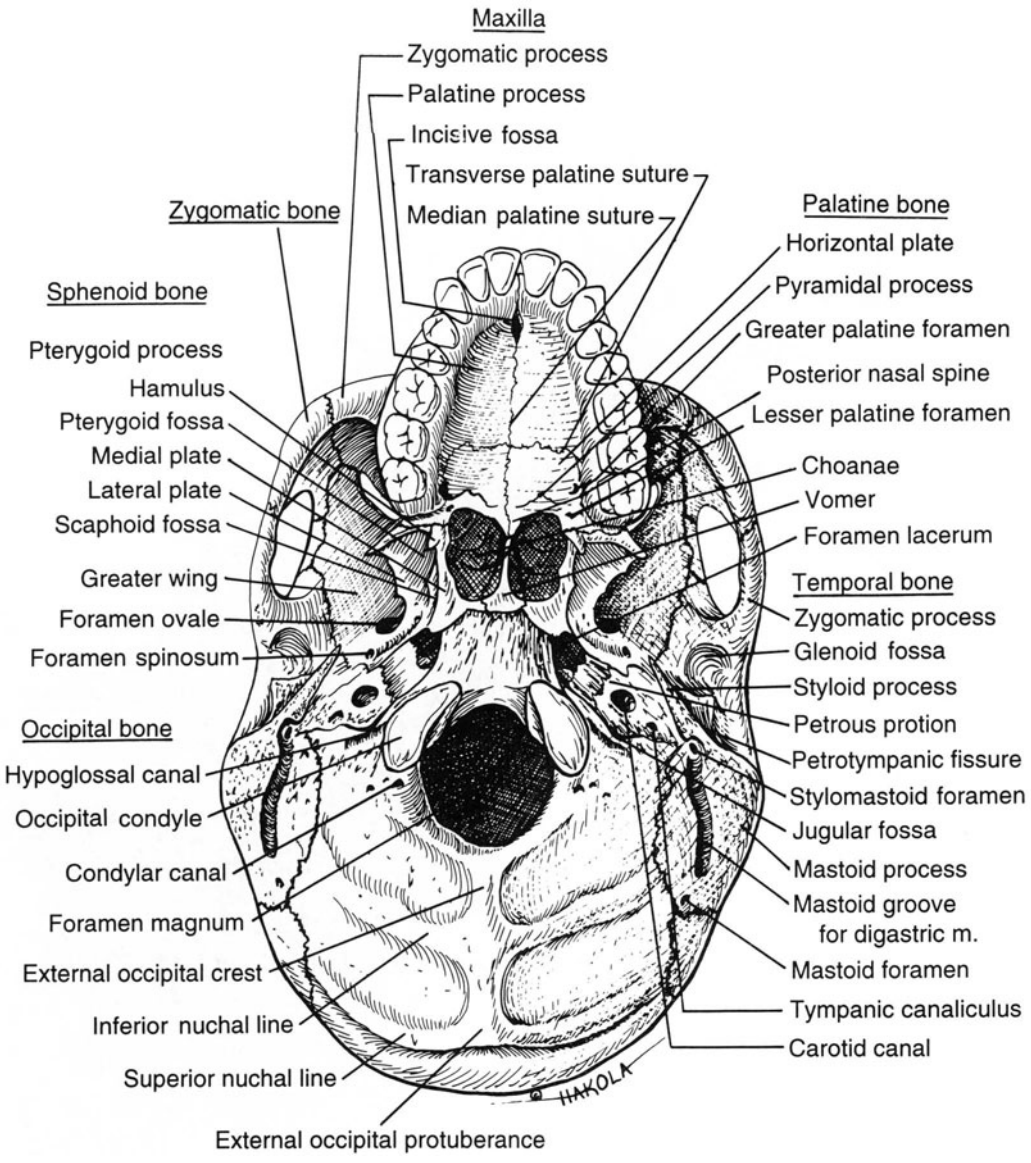


FIGURE 3.50. Inferior view of the cranial base.

The Cranial Bones

The distinctive anatomy of the cranial facial bones is best demonstrated in the following pages by Figures 3.51 to 3.56. References throughout the book may often be clarified by referring back to many of these figures.

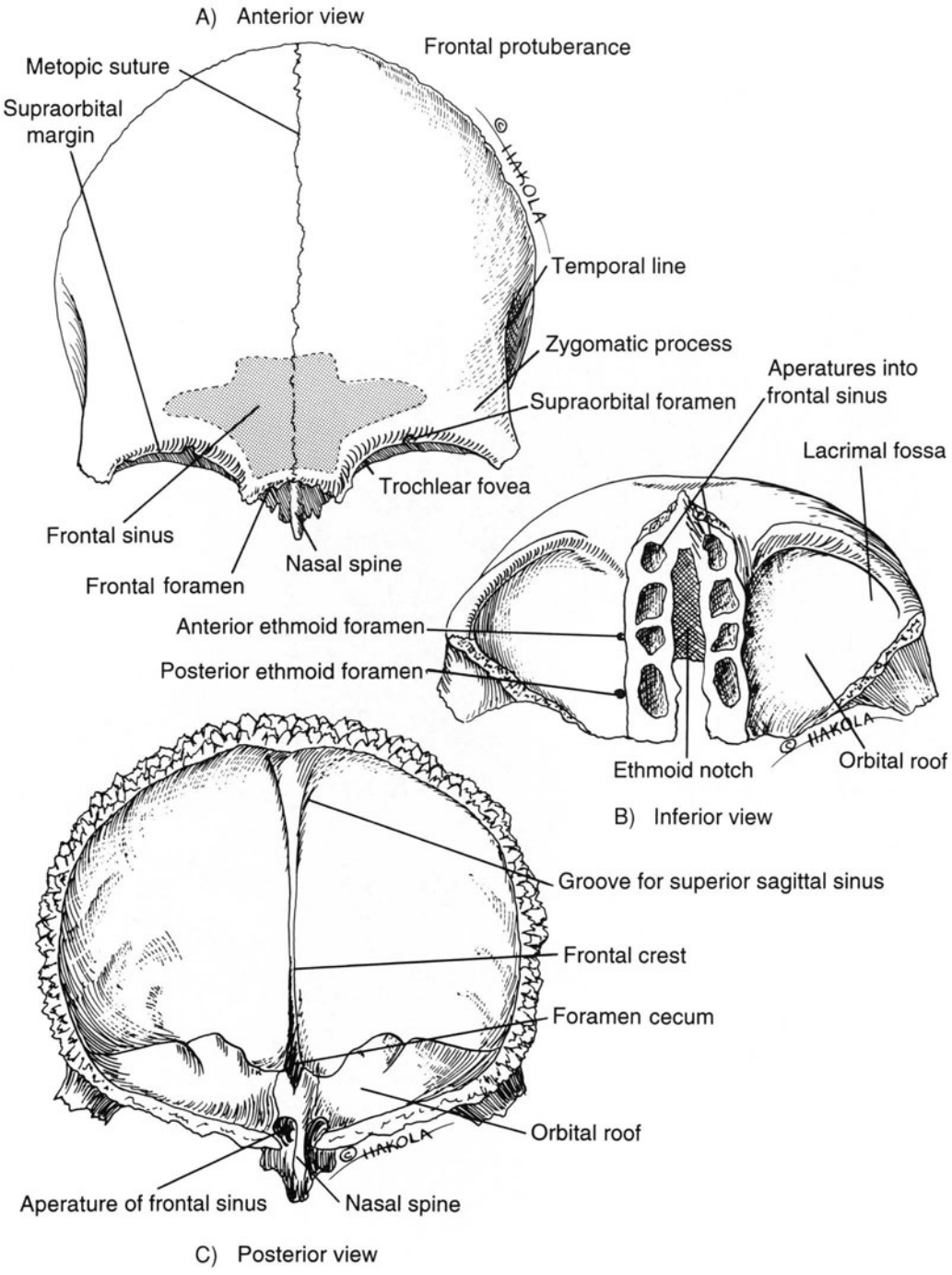


Figure 3.51. The frontal bone. (A) Anterior view; (B) inferior view; (C) posterior view.

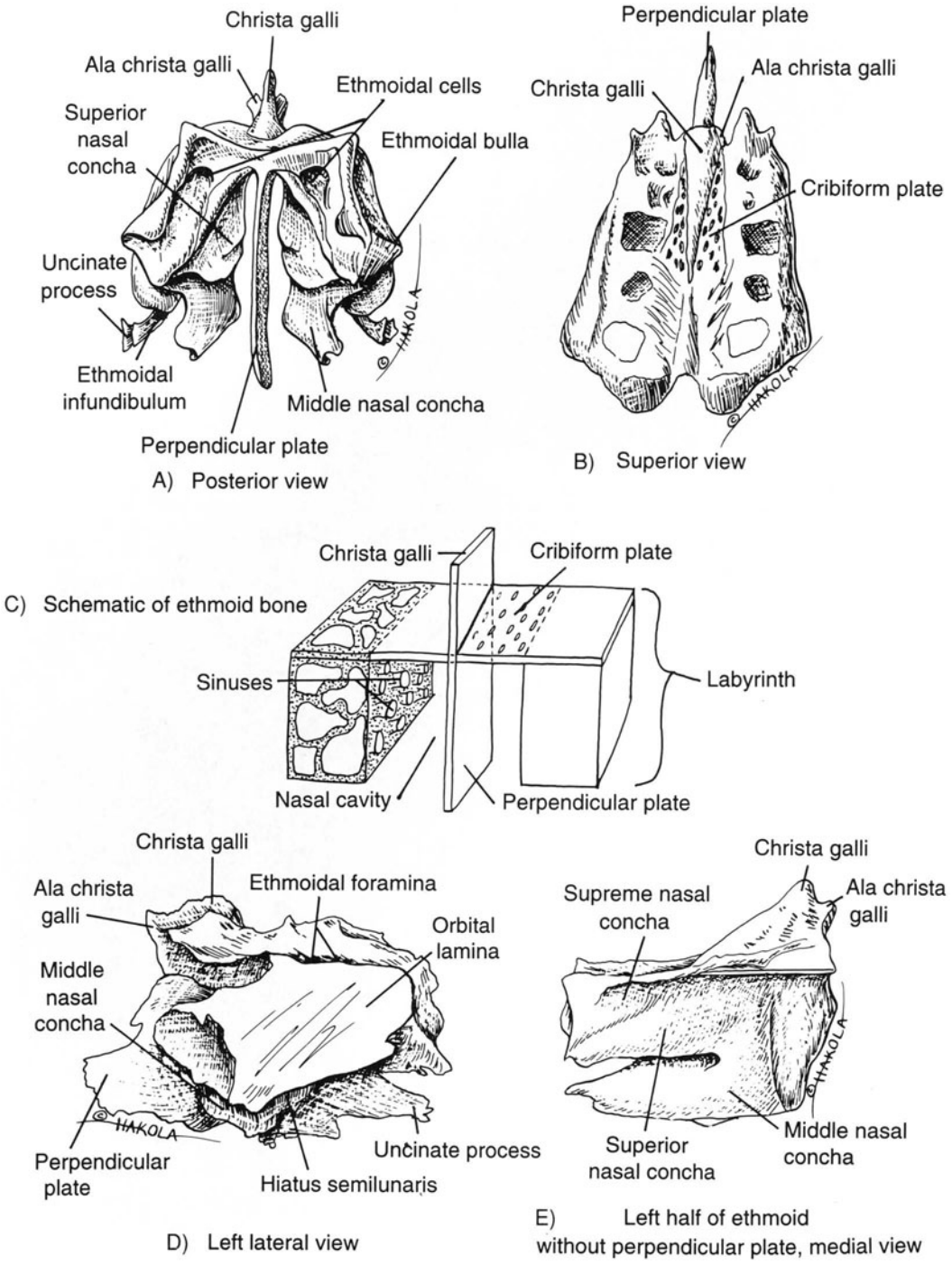


FIGURE 3.52. The ethmoid bone. (A) Posterior view; (B) superior view; (C) schematic of ethmoid bone; (D) left lateral view; (E) left half of ethmoid without perpendicular plate, medial view.

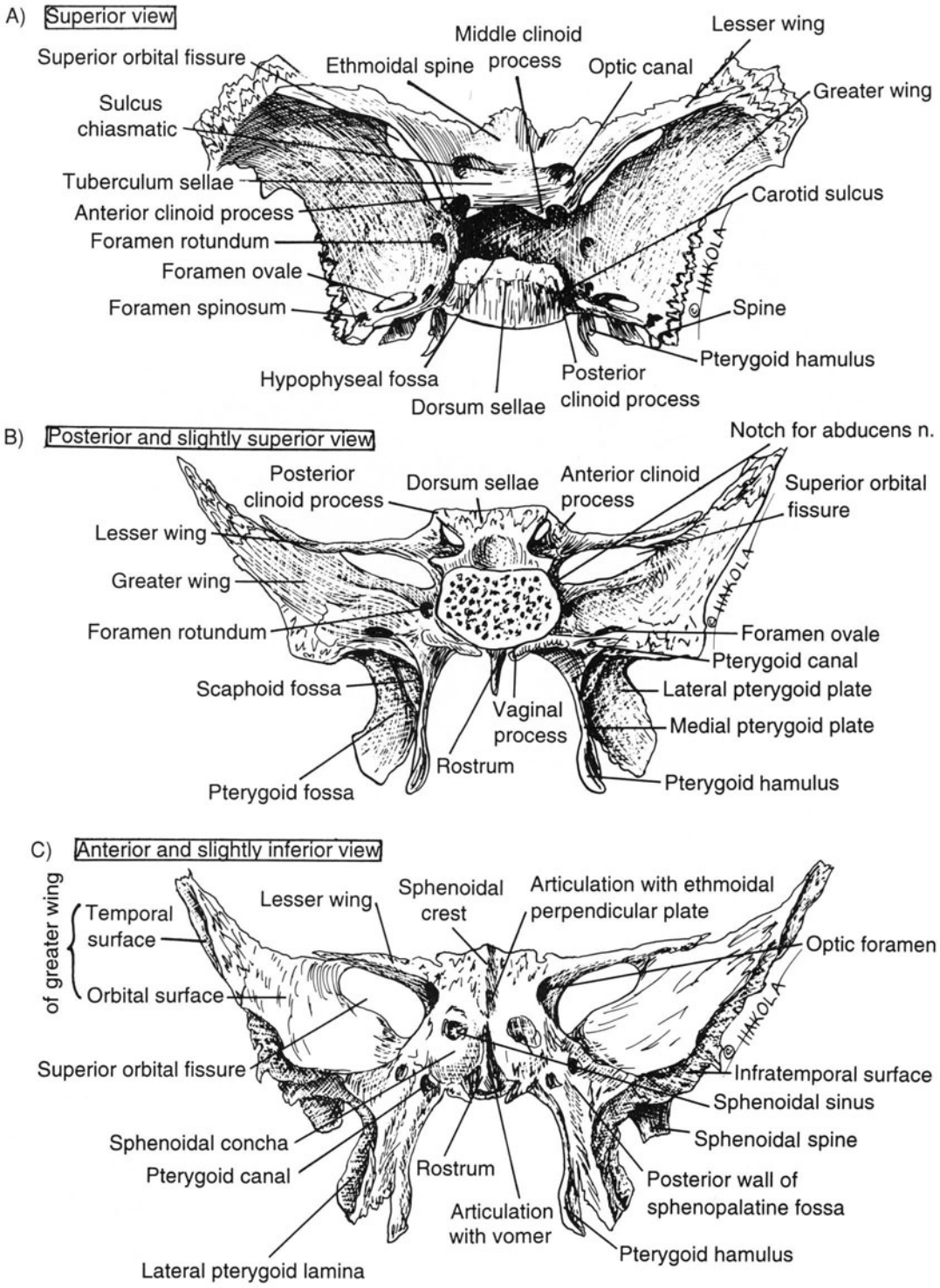


Figure 3.53. The sphenoid bone. (A) Superior view; (B) posterior and slightly superior view; (C) anterior and slightly inferior view.

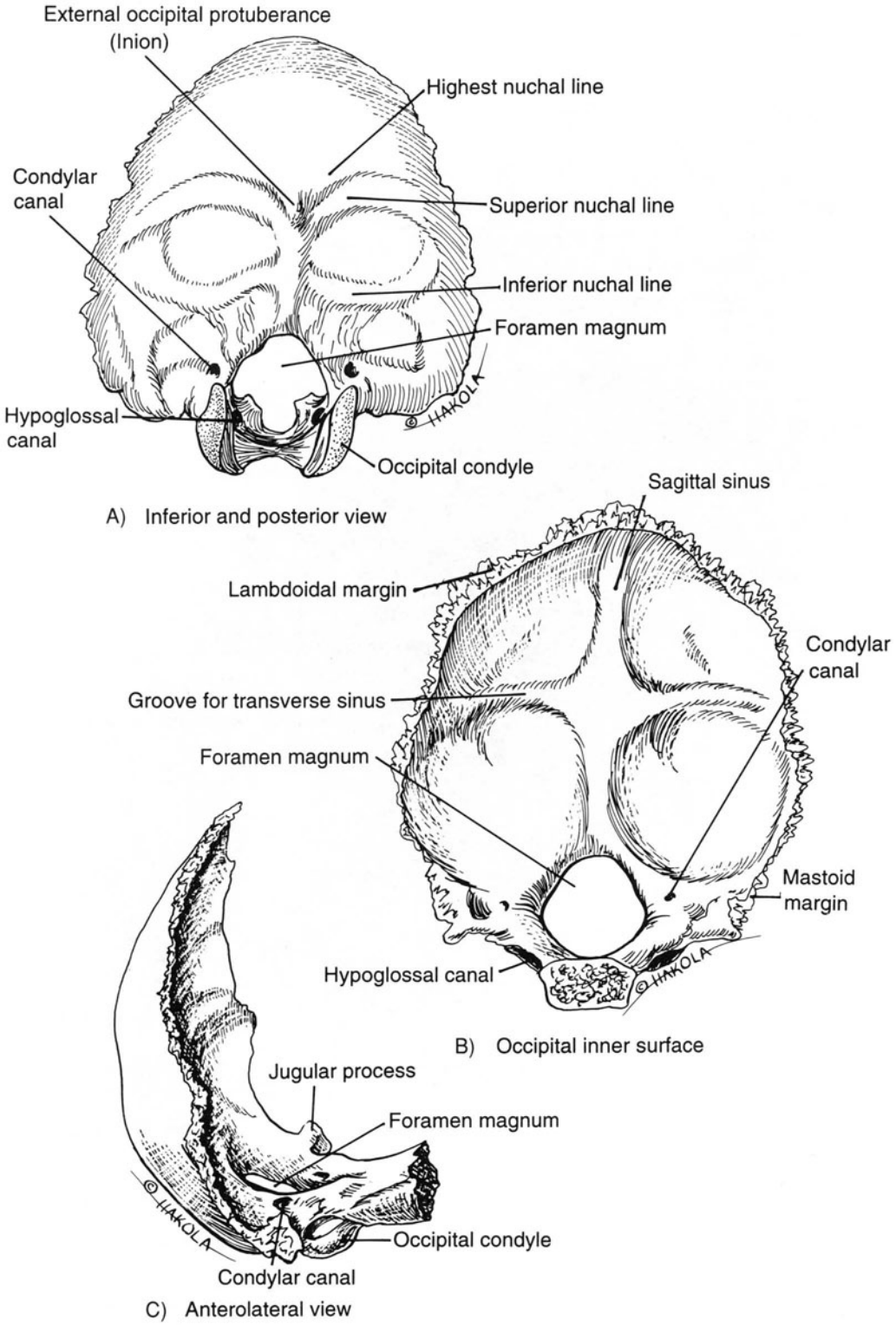


Figure 3.54. The occipital bone. (A) Inferior and posterior view; (B) occipital inner surface; (C) anterolateral view.

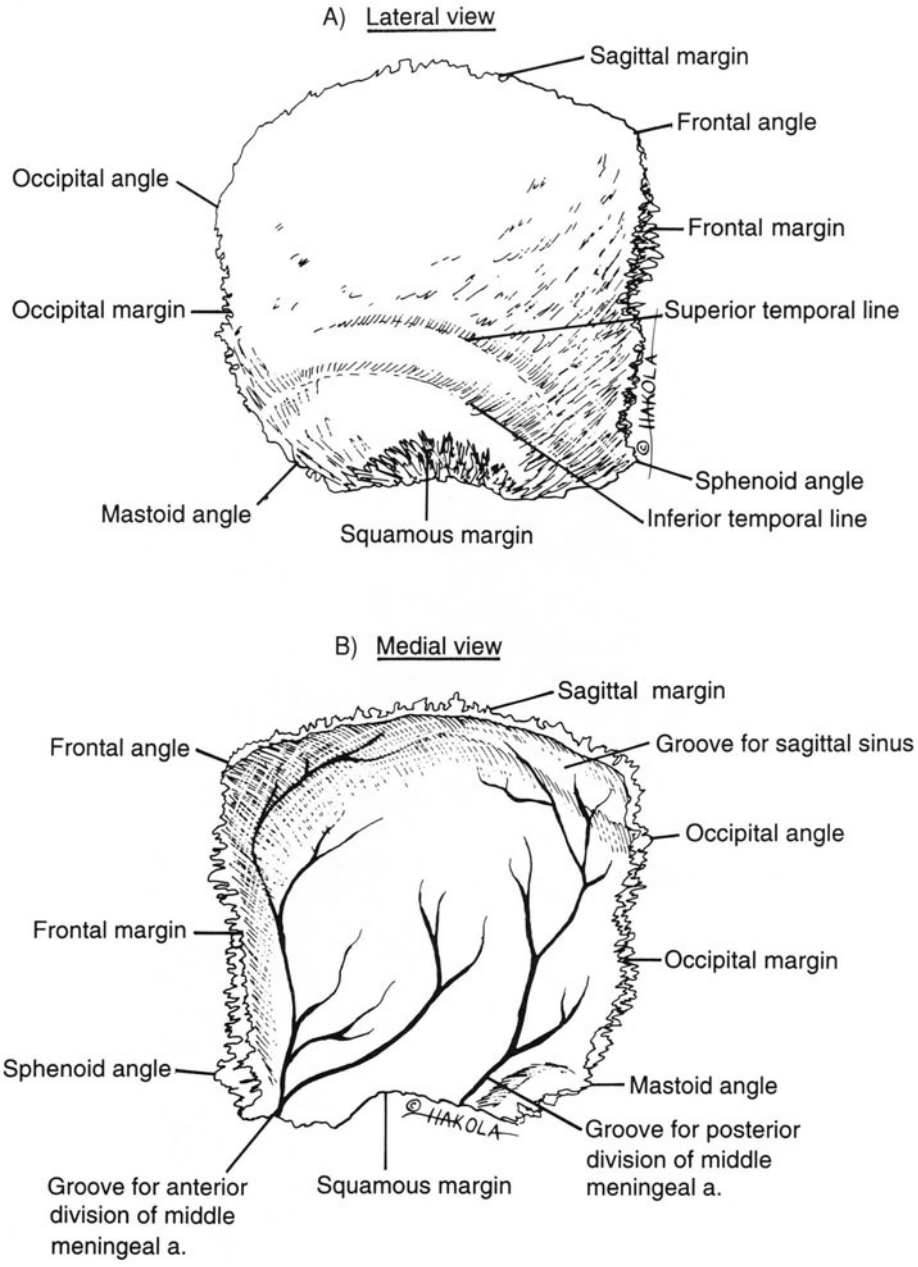
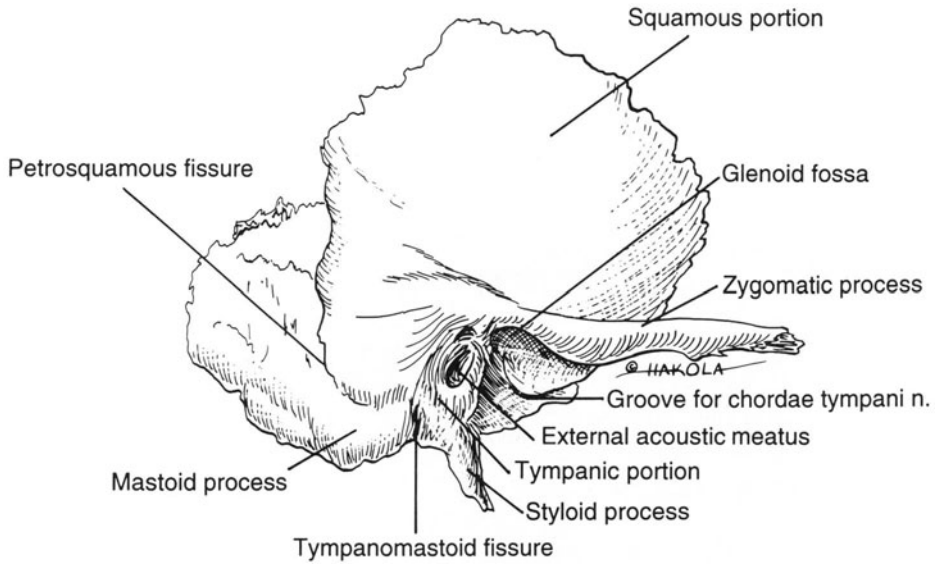


Figure 3.55. Parietal bone. (A) Lateral view; (B) medial view.

A) Lateral view



B) Medial view

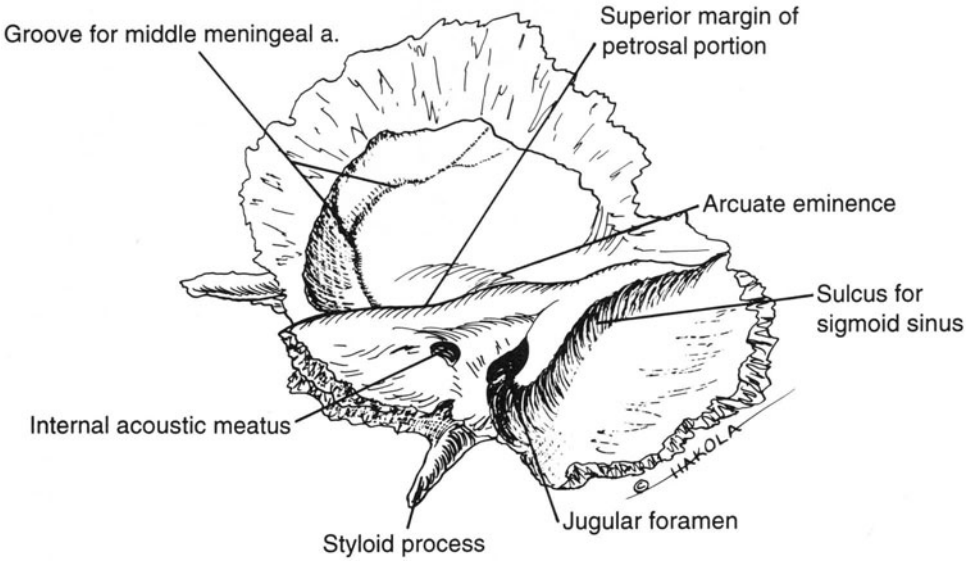


Figure 3.56. Temporal bone. (A) Lateral view; (B) medial view.

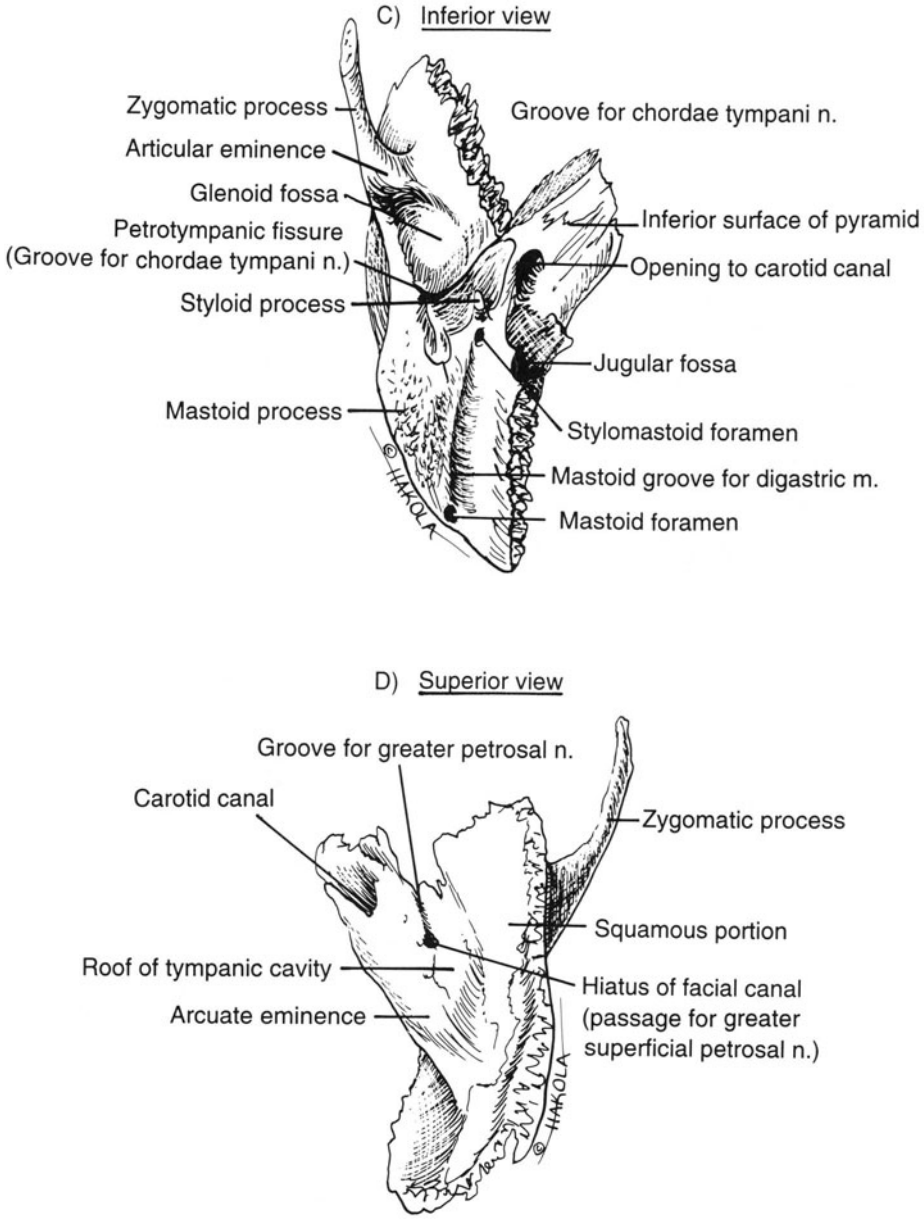


Figure 3.56. *Continued* (C) inferior view; (D) superior view.

The Facial Bones

Figures 3.57–3.64 illustrate various views of the individual facial bones. Many of the future discussions in this book can be clarified if the reader refers back to the drawings of these facial bones.

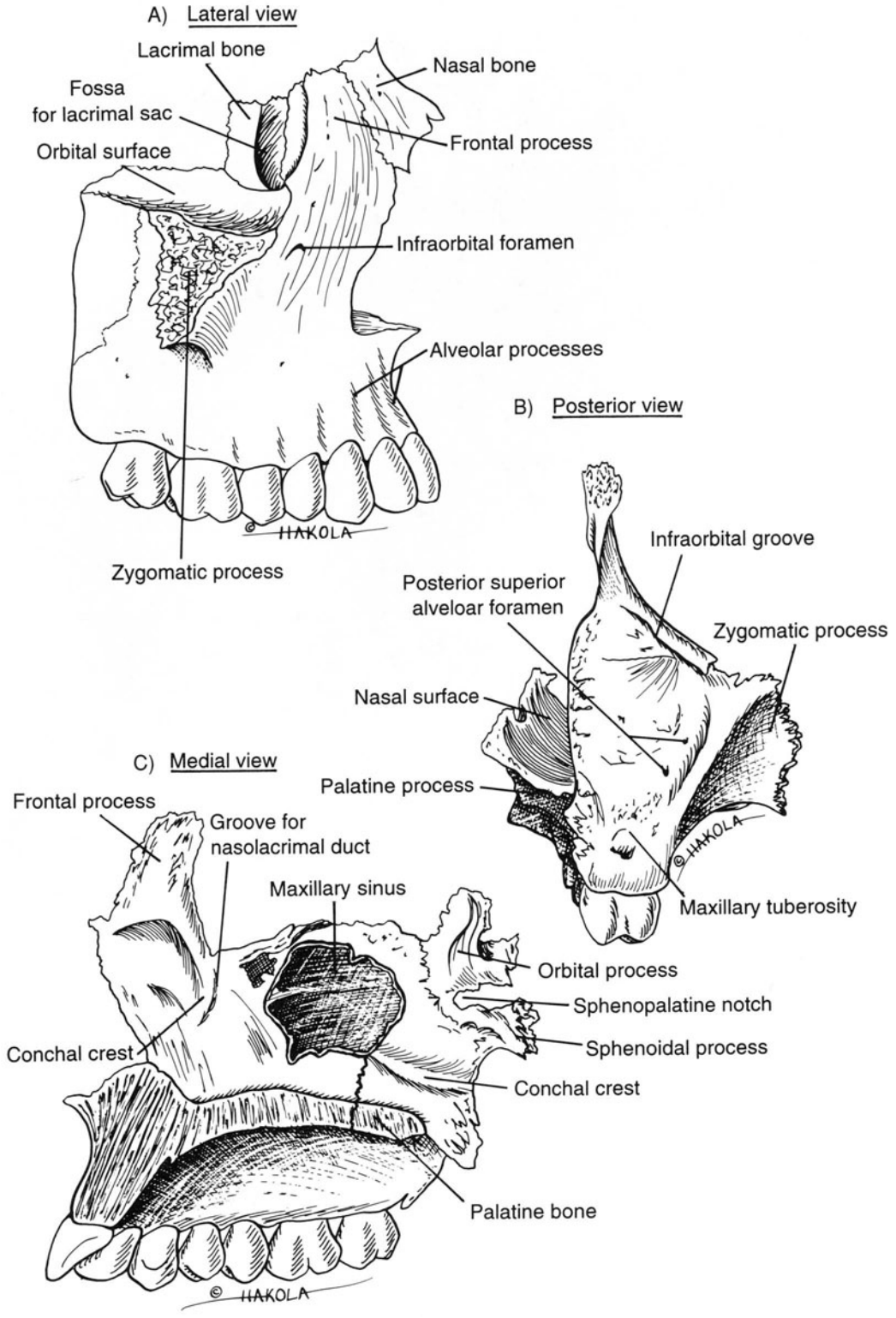


Figure 3.57. The maxilla. (A) Lateral view; (B) posterior view; (C) medial view.

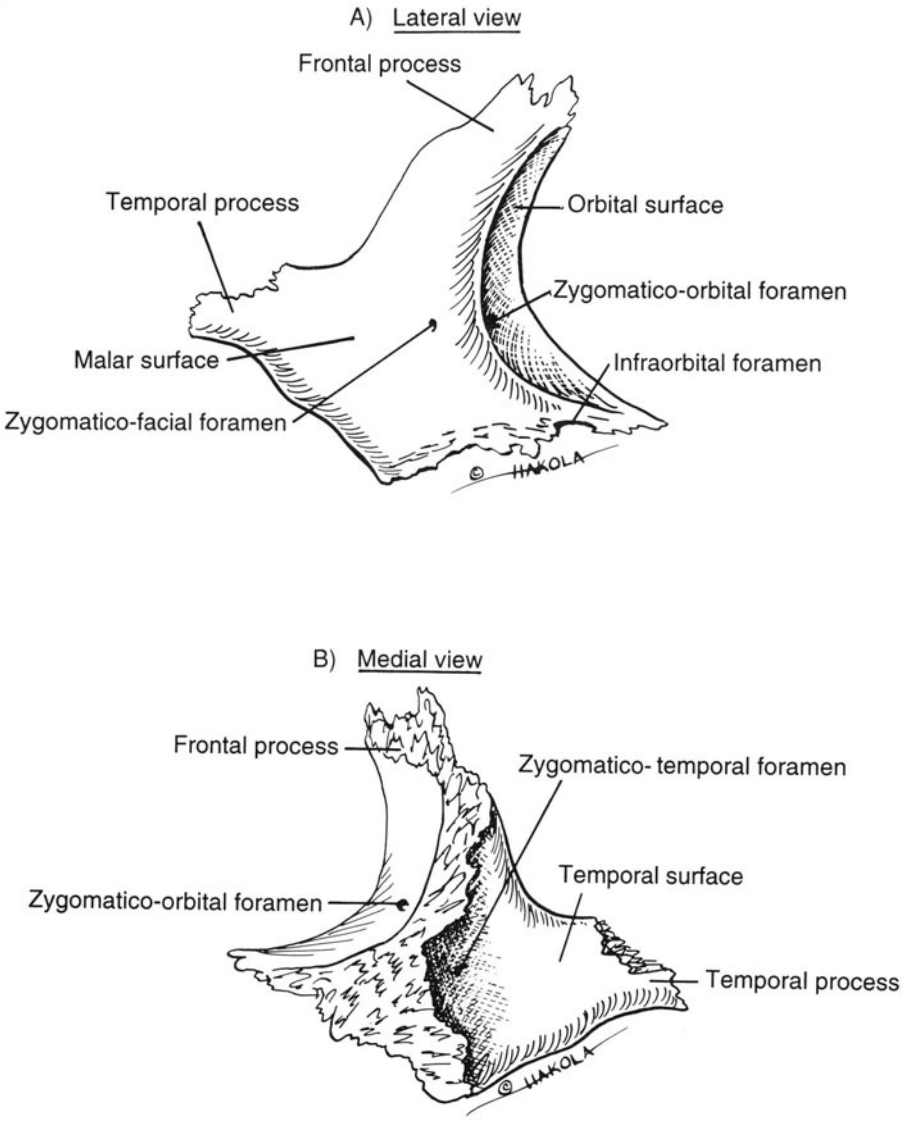


Figure 3.58. The zygoma. (A) Lateral view; (B) medial view.

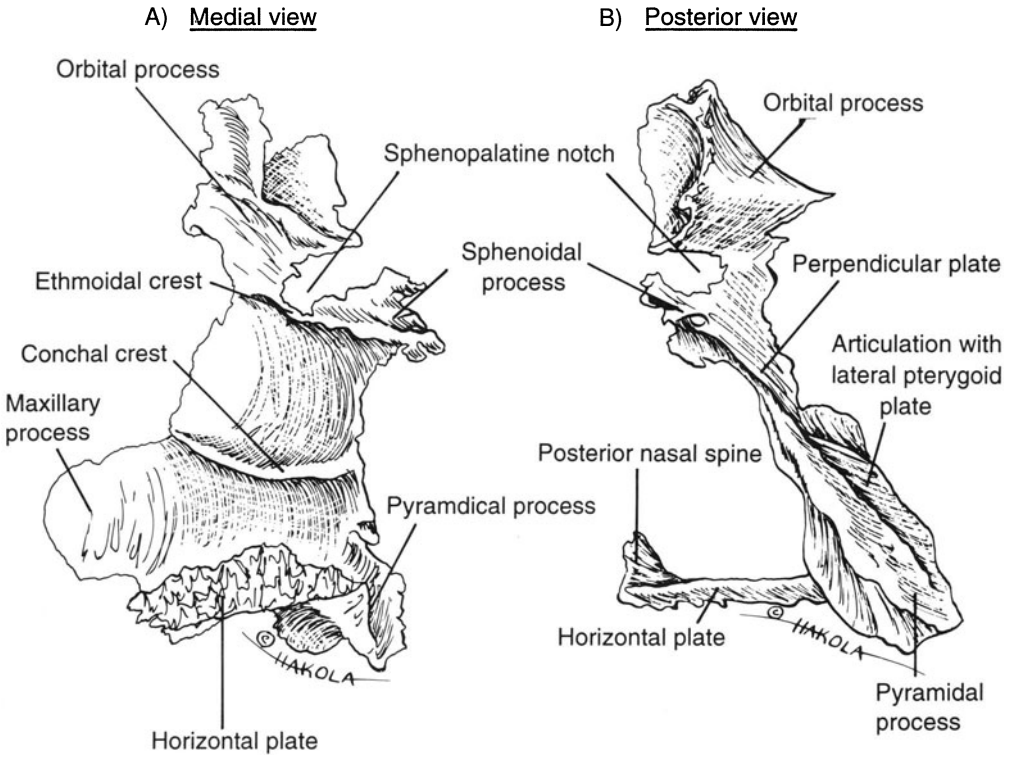


Figure 3.59. The right palatine bone. (A) Medial view; (B) posterior view.

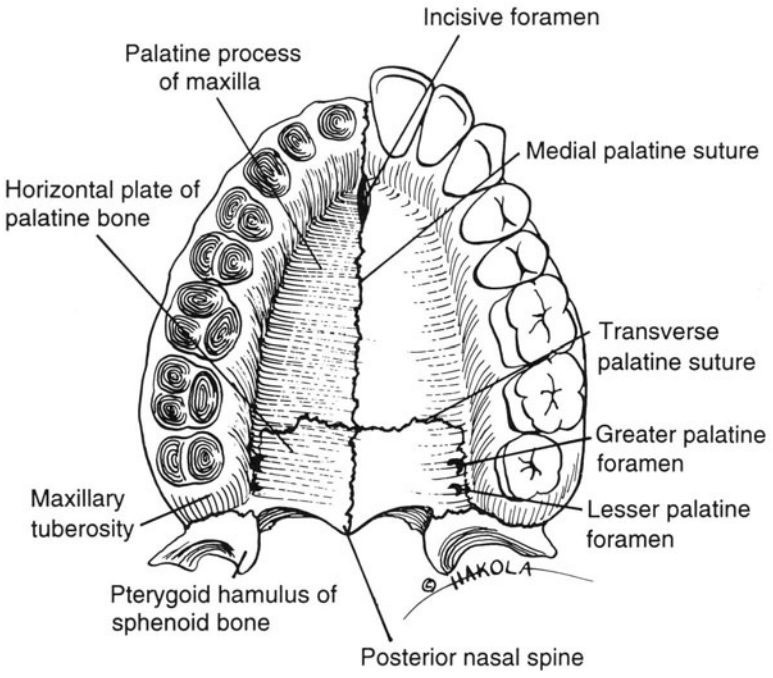


Figure 3.60. Inferior palatal view of maxillary-palatine junction.

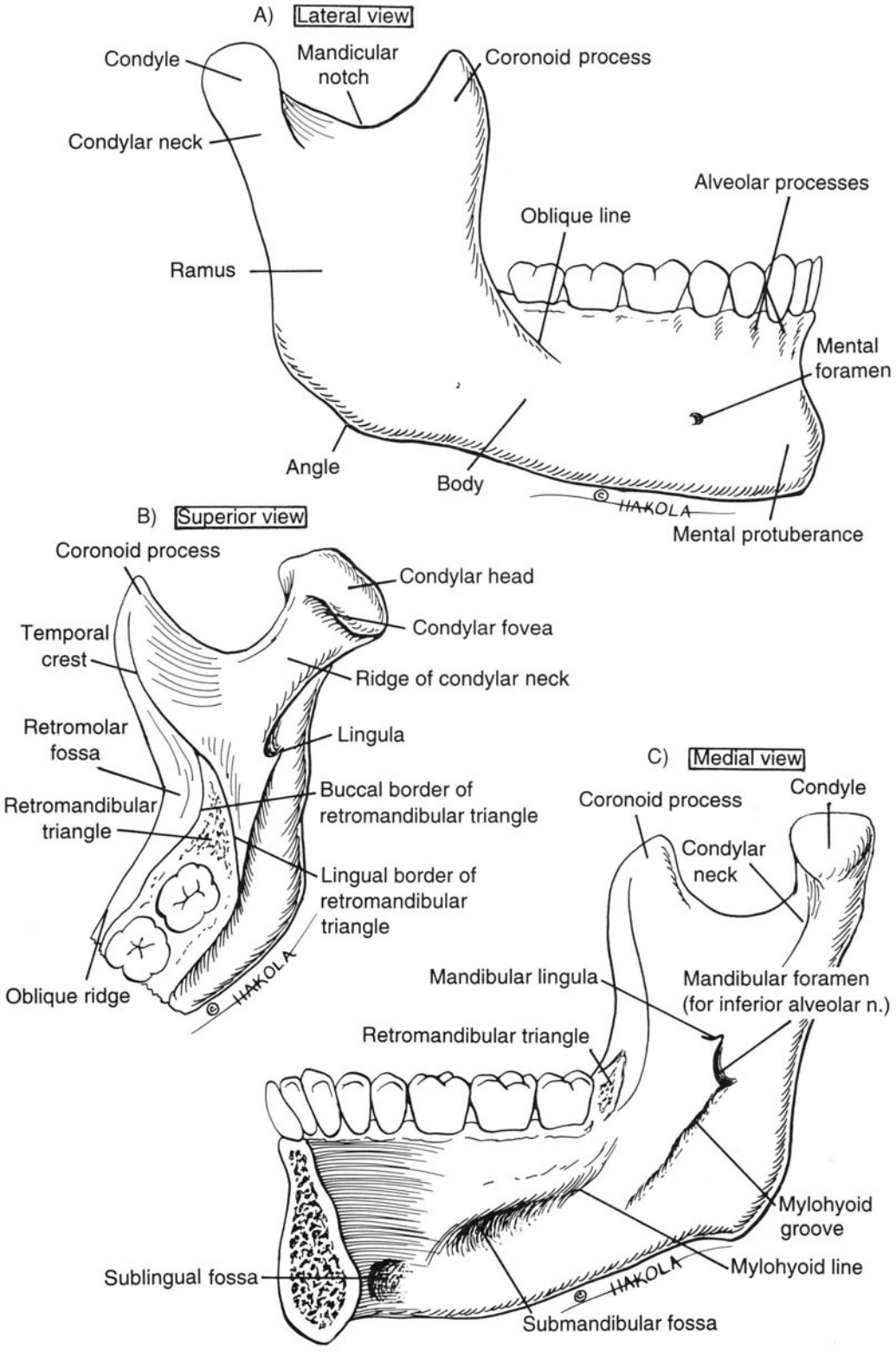


Figure 3.61. The mandible. (A) Lateral view; (B) superior view; (C) medial view.

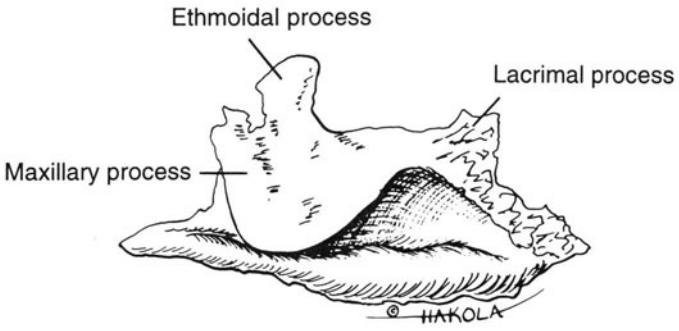


Figure 3.62. Lateral view left inferior concha.

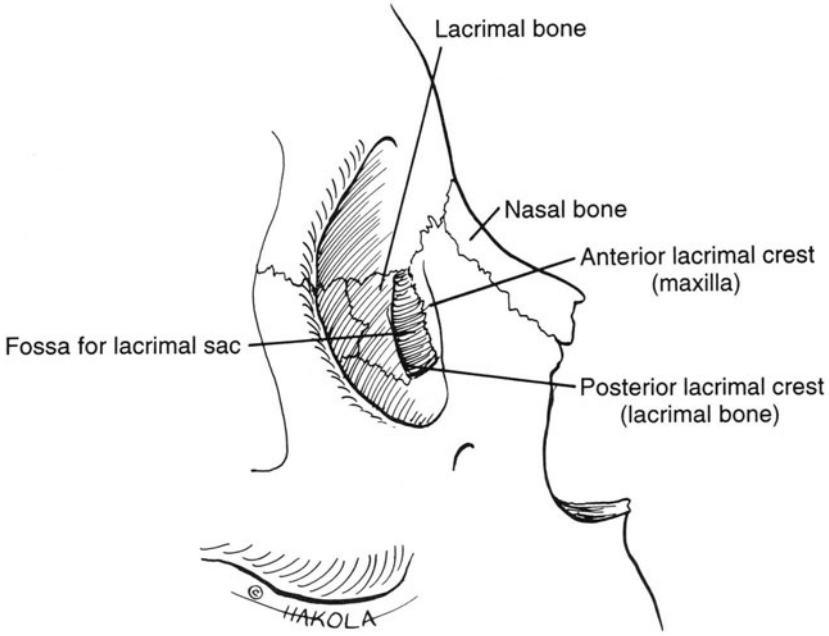


Figure 3.63. Lateral view lacrimal bone.

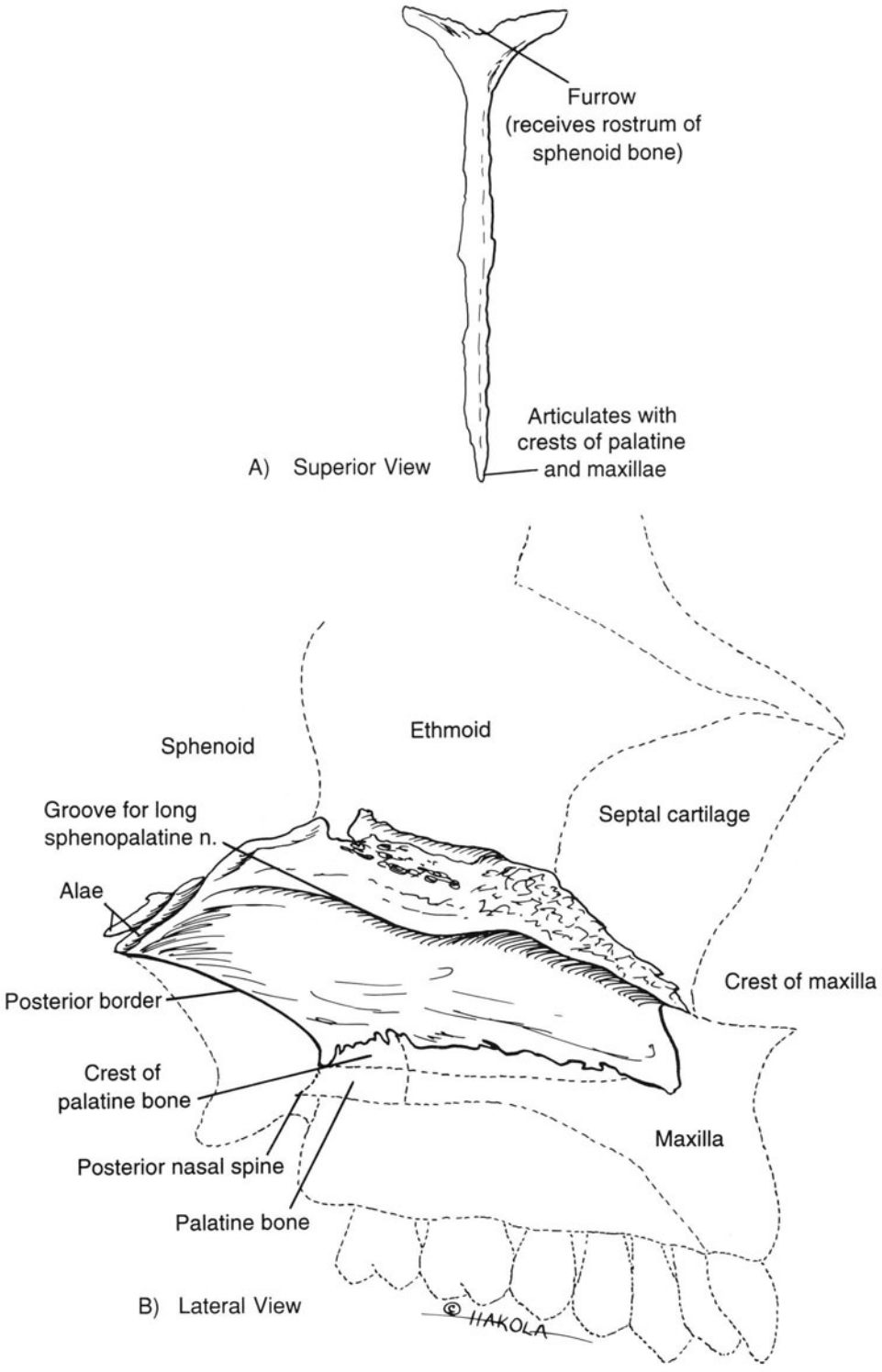


Figure 3.64. The vomer. (A) Superior view; (B) lateral view.

CHAPTER 4

Odontogenesis

Norman H. Rappaport

The histologic development of teeth is similar to the earliest stages of other organs that develop from the surface epithelium of the embryo; that is, epithelial proliferation occurs with subsequent invagination of mesenchymal tissue (Table 4.1). In the primitive oral cavity, the basal cuboidal layer of ectoderm (stratified squamous) is separated from the underlying mesenchyme by a basement membrane. The first evidence of tooth development occurs in utero at 6 to 7 weeks with a continuous cellular thickening, composed of proliferating epithelium and underlying mesenchymal compression, in the region of the future alveolar ridges (Fig. 4.1A). Epithelial invasion of the mesenchymal tissue creates the primary epithelial band, which follows the developing shape of the jaws. This primary epithelial band divides into the buccal and lingual bands of the vestibular lamina, the former becoming the vestibular sulcus and the latter becoming the dental lamina, the primordium of the ectodermal portion of the teeth (Fig. 4.1B).

Rounded epithelial swellings occurring at intervals along the length of the dental lamina represent the enamel organ of the developing primary, or deciduous, dentition (Table 4.2). The entire deciduous dentition is initiated by the second month in utero. The permanent teeth develop in two stages (Table 4.3). The successional dental lamina is the lingual extension of the primary dental lamina, with development taking place from the fifth month in utero (central incisor) to 10 months of age (secondary premolar) (Figs. 4.2 and 4.3). The accessional dental lamina is the distal extension of the primary dental lamina (Fig. 4.4). Evidence of an enamel organ for the developing permanent molars without deciduous predecessors is noted for the first molar at the fourth month in utero and after birth for the second and third molars. Additional information on the histological development of teeth can be found in Moors,[1] Arey,[2] Mjor and Fejerskov,[3] and Bhaskar.[4]

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Table 4.1. Summary of relations in tooth development.

Dental lamina
- Main lamina → Degenerates (including necks of enamel organs)
- Enamel organs of deciduous teeth
  - Outer enamel layer, Enamel pulp . . . → Pathway for enamel-forming materials → Dental cuticula
  - Inner enamel layer → Ameloblasts → Remnant / Enamel
  - Determines shape and size of crown
  - Organizes papilla and its odontoblast layer
  - Epithelial sheath → Determines shape and size of root; Organizes root papilla and its odontoblasts
- Free edge → Enamel organs of permanent teeth (except molars)
- Backward extension → Enamel organs of permanent molars . . . → Fates as above

Dental papilla
- Mesenchyme → Dental pulp / Fibrillar basis of dentine (?) → Dentine
- Odontoblast layer → Calcifies dentine (?) / Dentinal fibers (of Tomes)

Dental sac
- Outer region → Osteoblasts → Alveolar bone of jaw
- Middle region → Fibroblasts → Periodontal membrane
- Inner region → Cementoblasts → Cementum

→ Alveolar wall

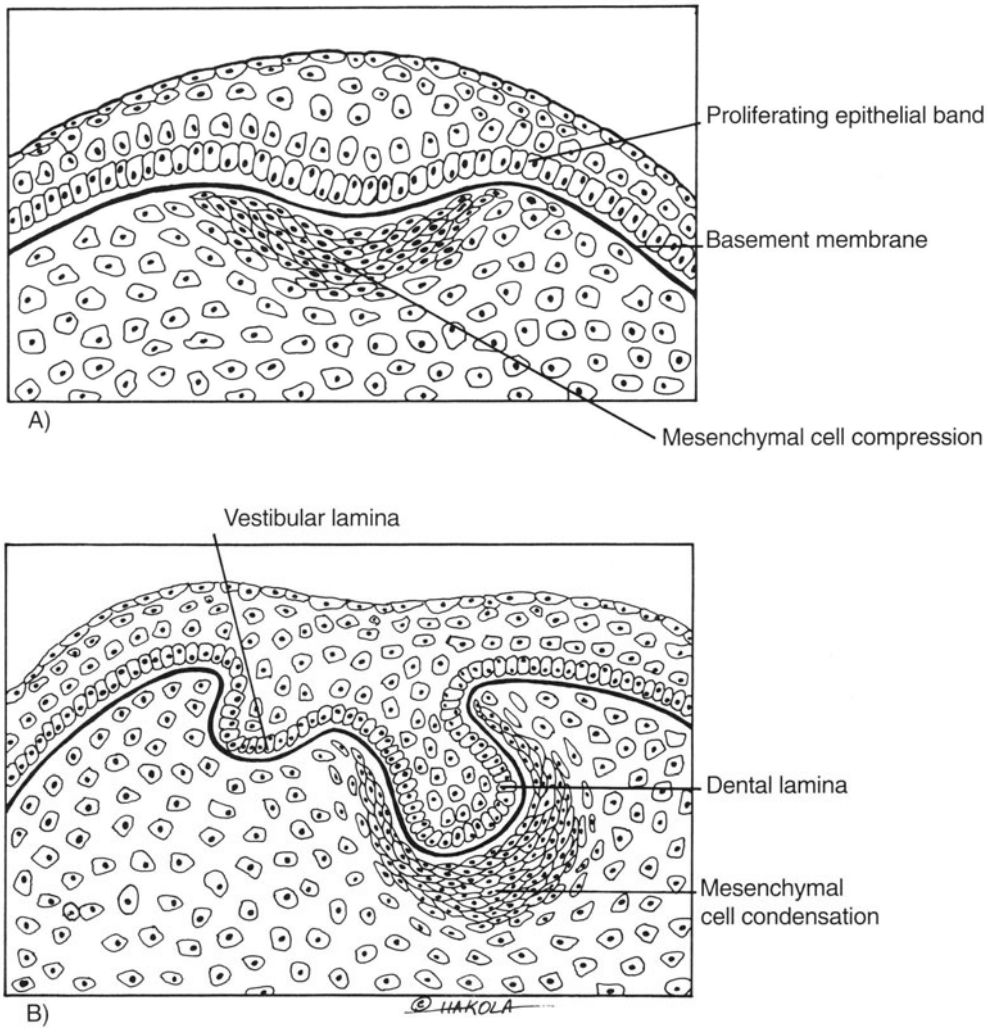


Figure 4.1. Schematic diagrams showing early stages in human tooth development. (A) Shows formation of the primary epithelial band in relation to an underlying mesenchymal cell compression. (B) Shows the dental lamina growing into the mesenchymal cell condensation and the vestibular lamina growing bucally.

Table 4.2. The development and eruption of the deciduous teeth.

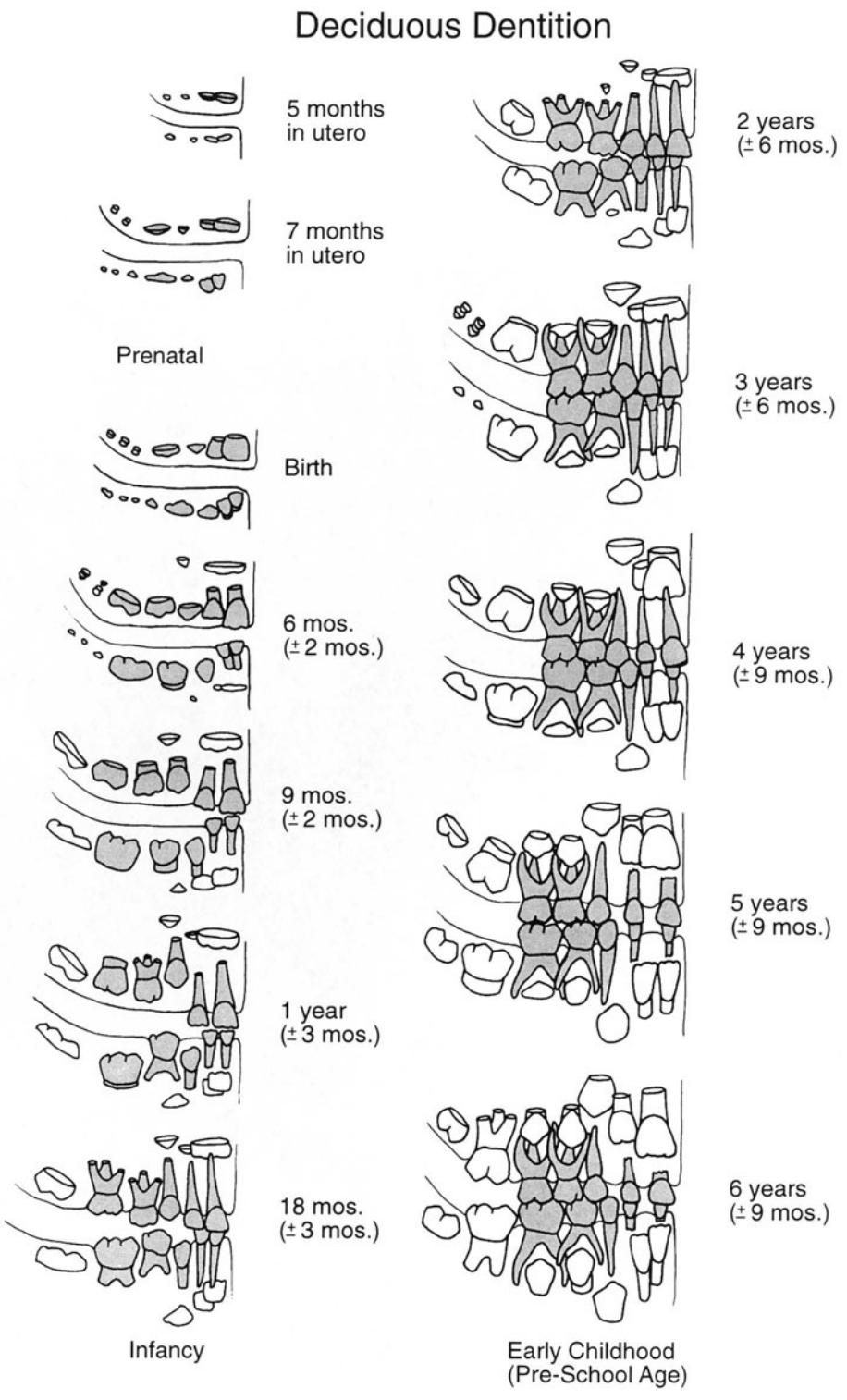
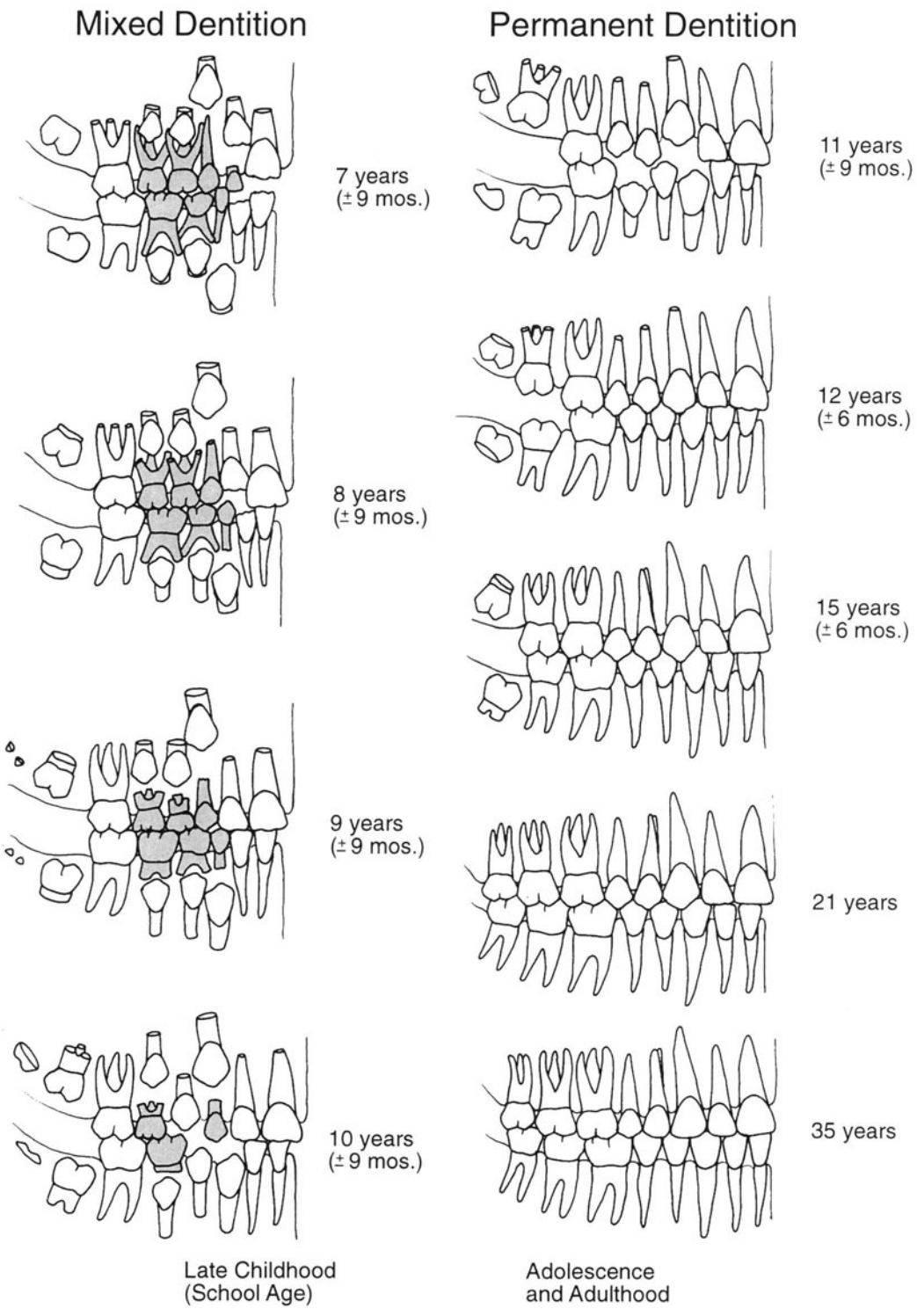


Table 4.3. The development and eruption of the mixed and permanent dentition.



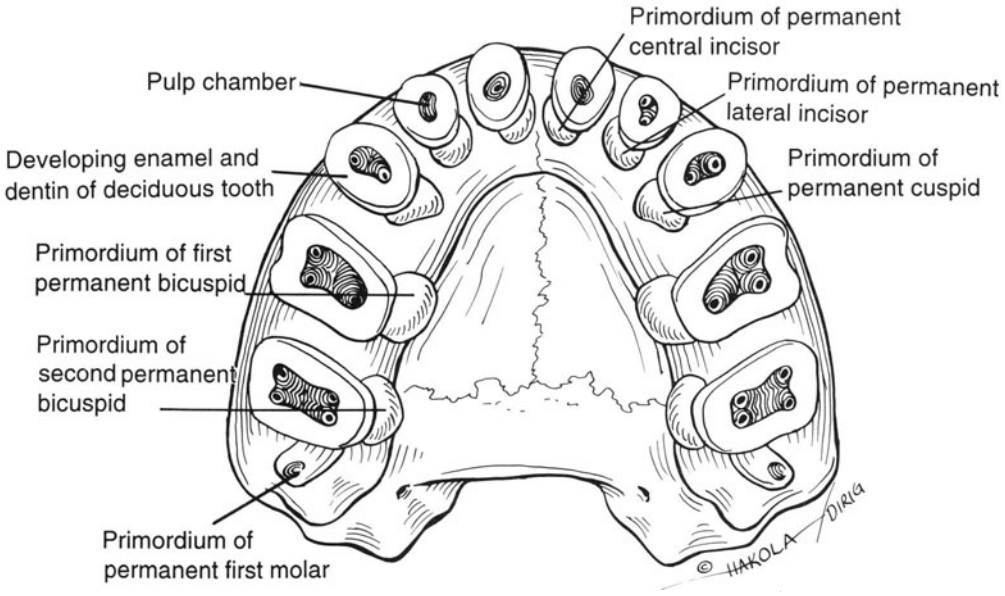


FIGURE 4.2. Primordia of permanent teeth are seen as thickenings of dental lamina on lingual side of each tooth germ. Distal extension of dental lamina with primordium of first molar. Approximately a 4-month embryo.

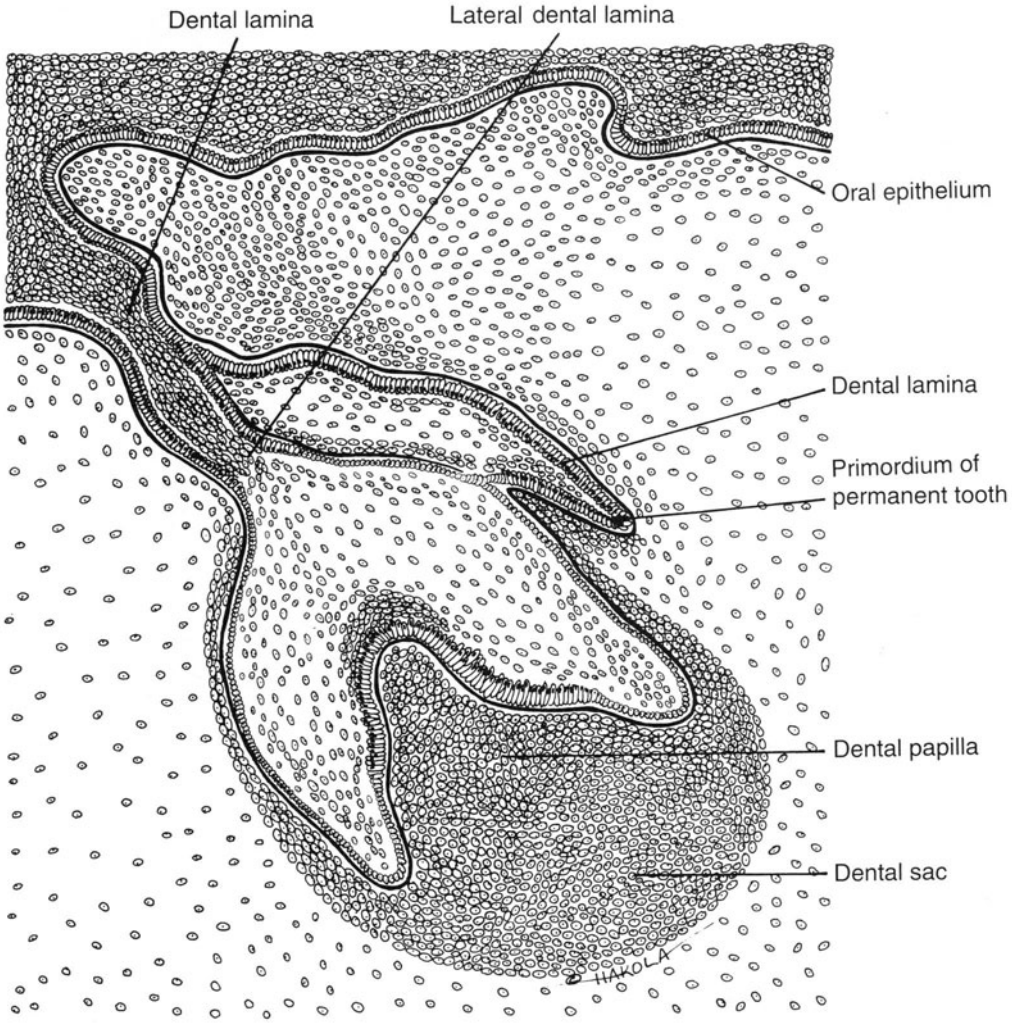


FIGURE 4.3. Labial-lingual section of developing primary tooth and primordium of permanent tooth.

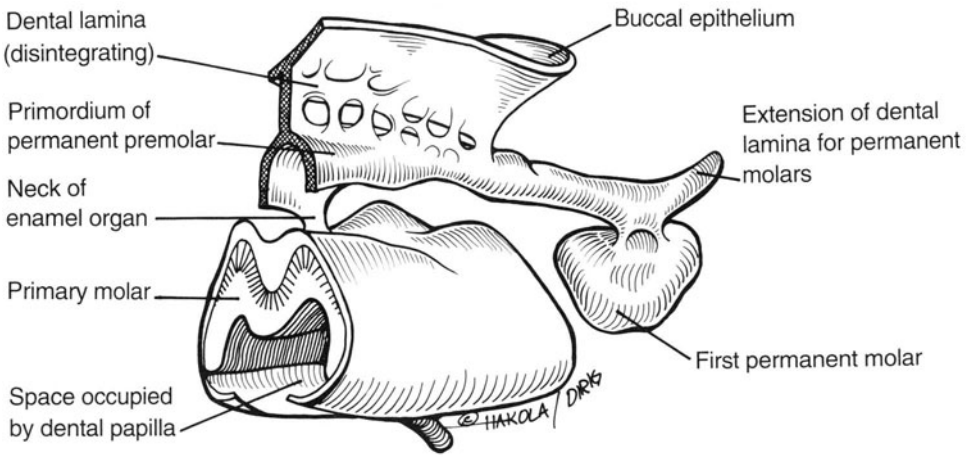


Figure 4.4. Permanent molar developing from the backward extension of the dental lamina.

Crown Development

The enamel organ, derived from ectodermal elements, constitutes the tooth bud (bud stage); the structure consists of low, columnar, tightly packed cells at the periphery surrounding a central core of more loosely arranged polygonal cells (Fig. 4.5A). Mesenchymal cells, thought to derive from neural crest cells, condense at the proliferating tip of the enamel organ, giving rise to the dental papilla and dental sac.

As the epithelial cells of the enamel organ proliferate further, the mesenchymal cells are surrounded in a follicle-like arrangement (cap stage). The convex surface of the enamel organ is lined by the outer enamel epithelium and the concave surface by the inner enamel epithelium; the polygonal epithelial cells at the core become the stellate reticulum (Fig. 4.5B). An additional condensation of cells located centrally at the edge of the inner enamel epithelium forms the enamel knot with its extension, the enamel cord, which may serve as a nidus of dividing cells of the enamel organ.

As invagination continues, the enamel organ continues to enlarge into a bell-shaped structure (bell stage). The outer enamel epithelium remains composed of low cuboidal cells separated from the surrounding mesenchymal cells of the dental sac by a basement membrane (Fig. 4.6). The polygonal cells of the stellate reticulum become increasingly separated as they accumulate intercellular glucosaminoglycans, and the stratum intermedium, essential for enamel formation, divides the stellate reticulum from the inner enamel epithelium. The cells of the inner enamel epithelium are columnar and are set apart from the peripheral cells of the dental papilla by a basement membrane in a zone that is virtually cell free. Long, slender cytoplasmic extensions of the mesenchymal cells approach the epithelium across this zone.

Movement of the odontoblasts away from the basement membrane precedes dentin formation. The cell-free zone beneath the basement membrane contains the cytoplasmic extensions from the odontoblasts, together with perpendicularly oriented fibrils which, when surrounded by ground substance, form predentin. As the predentin mineralizes, the basement membrane disappears, and the cytoplasmic extensions of the odontoblasts encounter the inner enamel epithelium, differentiating into ameloblasts with subsequent enamel deposition (Fig. 4.7A and 4.7B).

Tooth shape is determined by interaction among tissue elements. Hard tissue formation begins at the future enamel-dentin junction of the incisal edge or cusp tip. Cytodifferentiation is maximal prior to matrix secretion. The mesenchymal cells become polarized, tall odontoblasts; the inner enamel epithelial cells also polarize as they differentiate into ameloblasts. Within the cell-free zone, membrane-bound vesicles may be responsible for the delivery of "developmental information." Interaction within this zone is crucial because mesenchymal differentiation into odontoblasts cannot occur unless the cells are in close proximity to the inner enamel epithelium. Enamel formation takes place only following extracellular matrix formation from the odontoblasts.

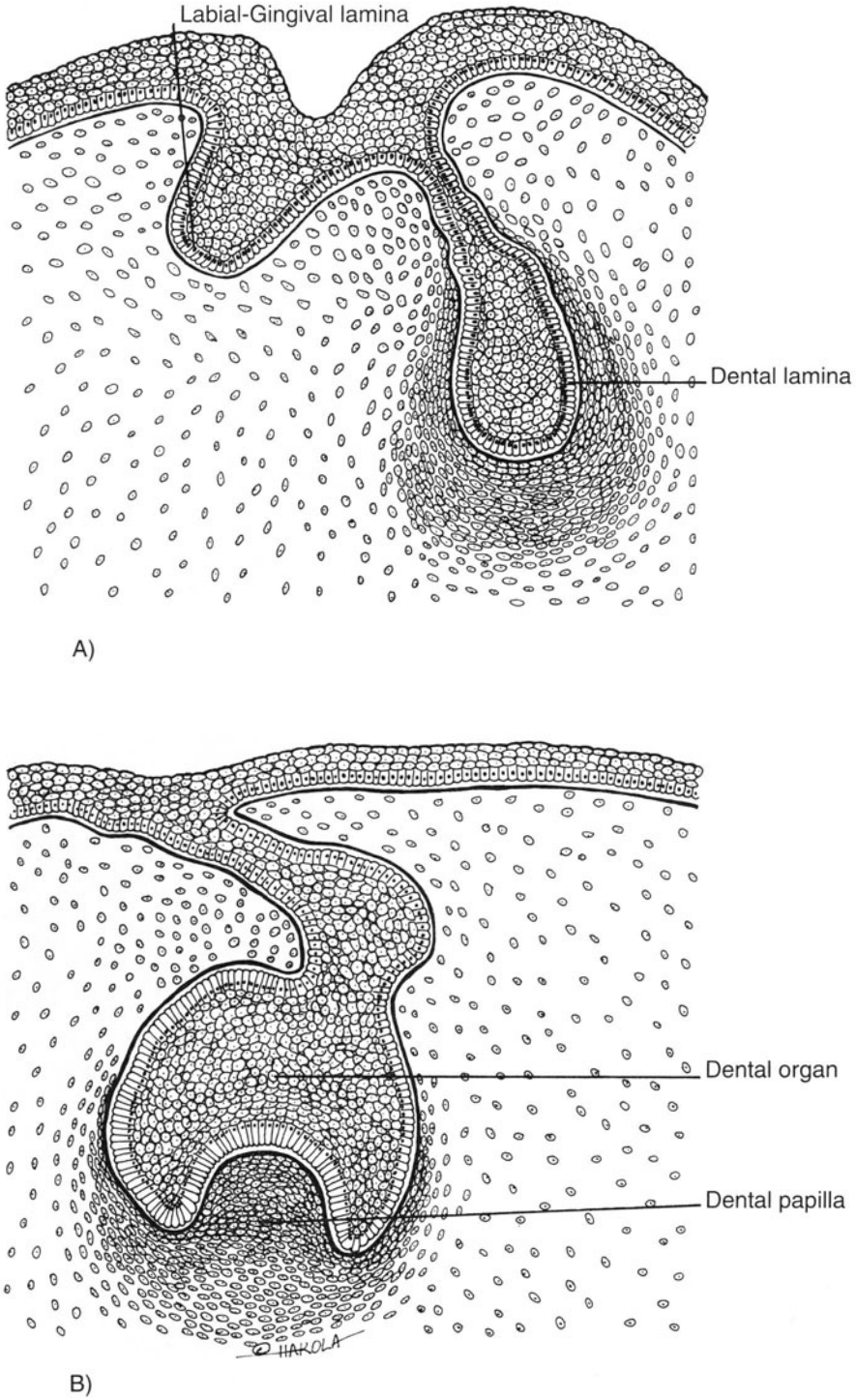


Figure 4.5. (A) Epithelial bud forms at the end of the dental lamina—"bud stage." (B) The ectodermal cells proliferate and form a cap on top of mesenchymal cells—"cap stage."

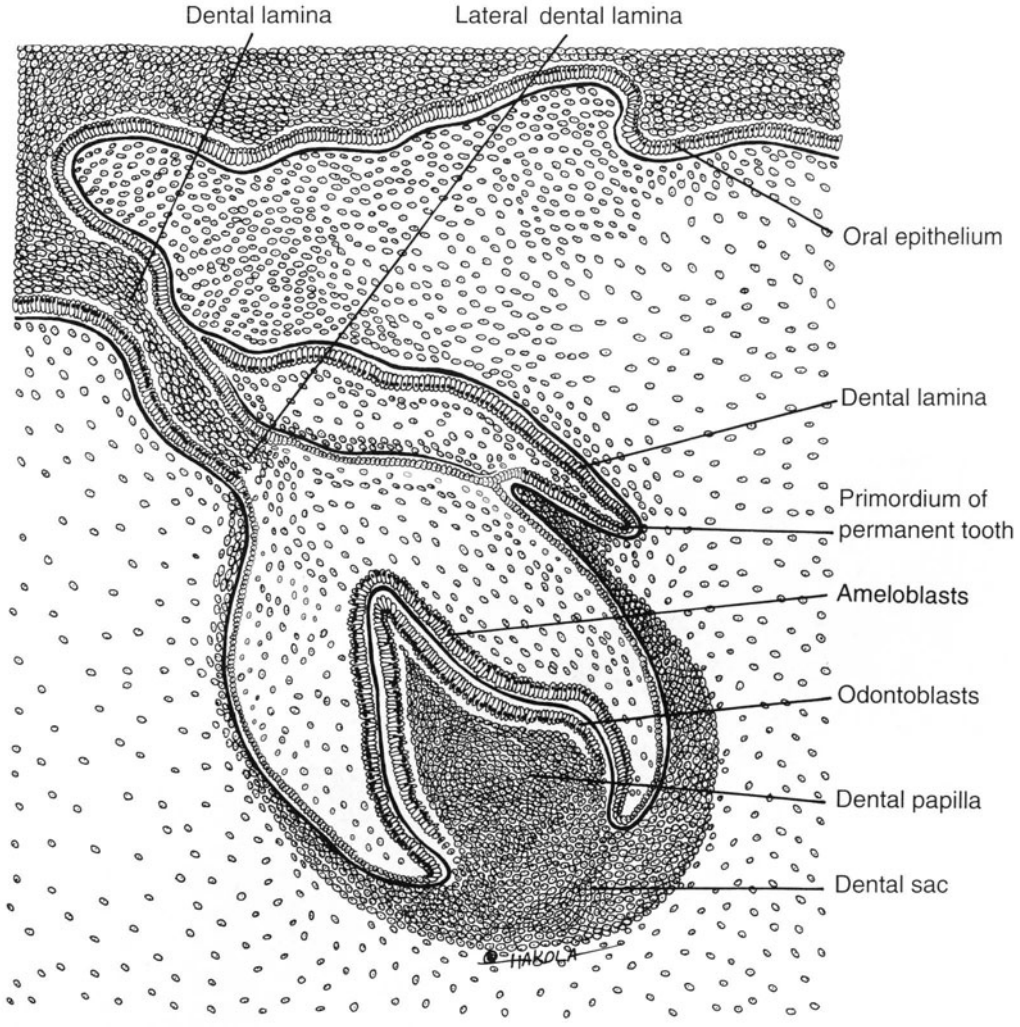


Figure 4.6. Further growth resulting in a bell-shaped enamel organ—the "bell stage" of tooth development.

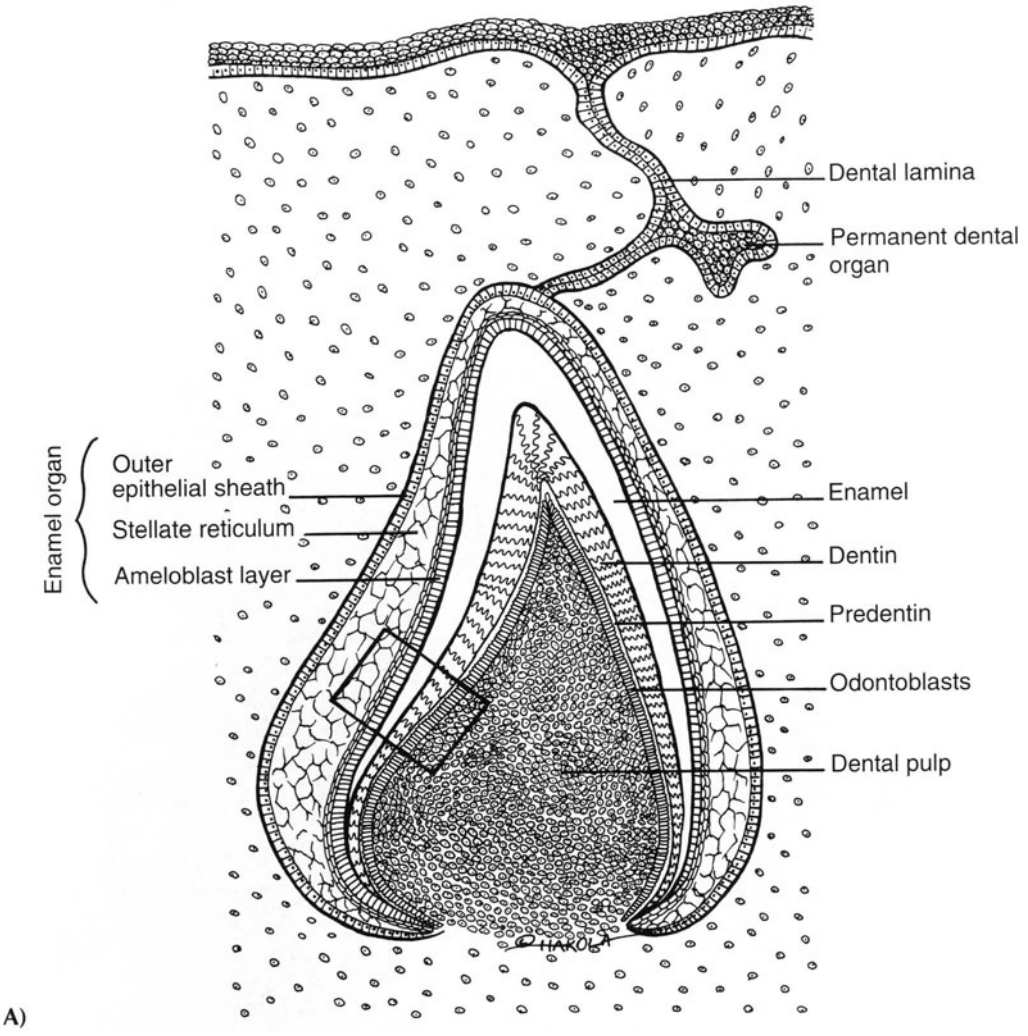


Figure 4.7. (A) Cell differentiation (epithelial and mesenchymal) in early tooth crown formation.

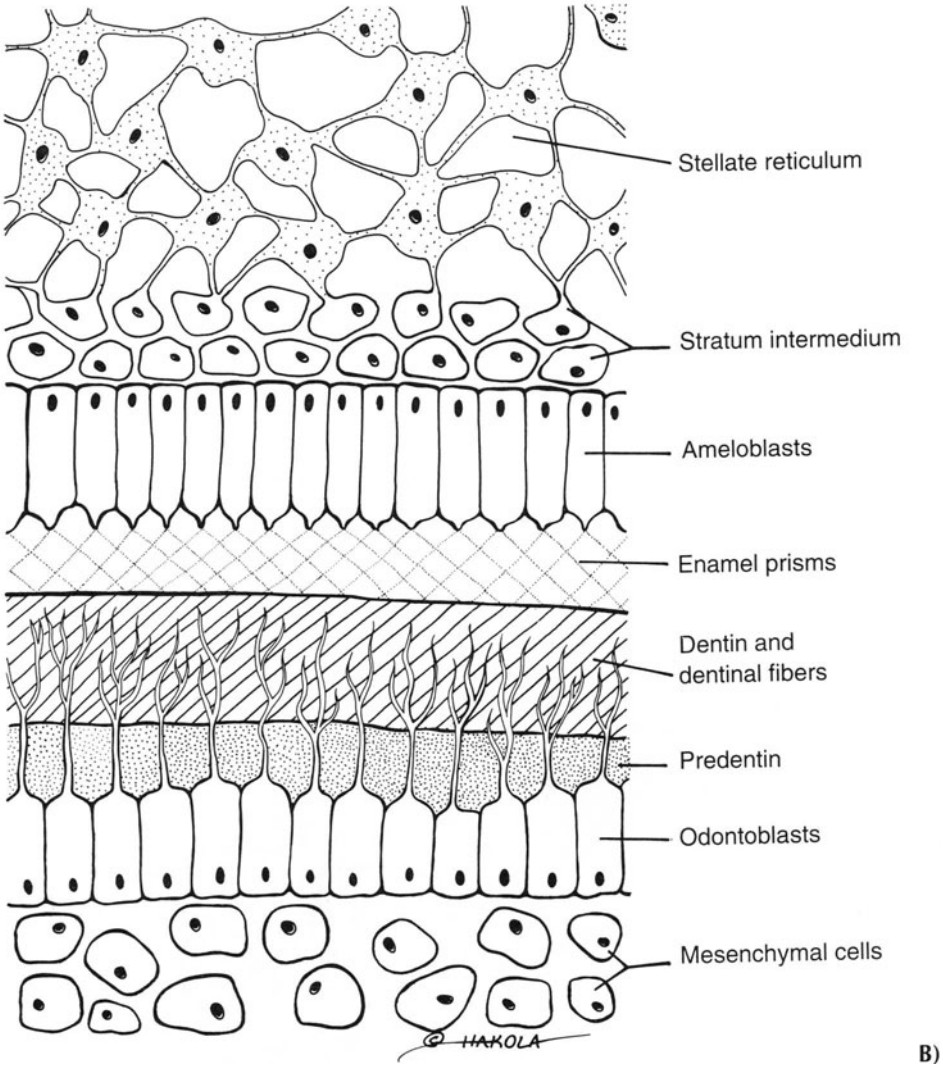


Figure 4.7. *Continued* (B) Schematic enlargement of boxed area seen in Fig. 4.4A.

Root Development

Cell proliferation from the enamel organ extends apically after enamel and dentin formation reach the eventual cementoenamel junction. The inner and outer enamel epithelia develop into Hertwig's root sheath, which is responsible for root formation (Fig. 4.8A). There is no stellate reticulum between the enamel epithelial layers at this level; the inner enamel epithelial cells differentiate into odontoblasts rather than ameloblasts. Predentin

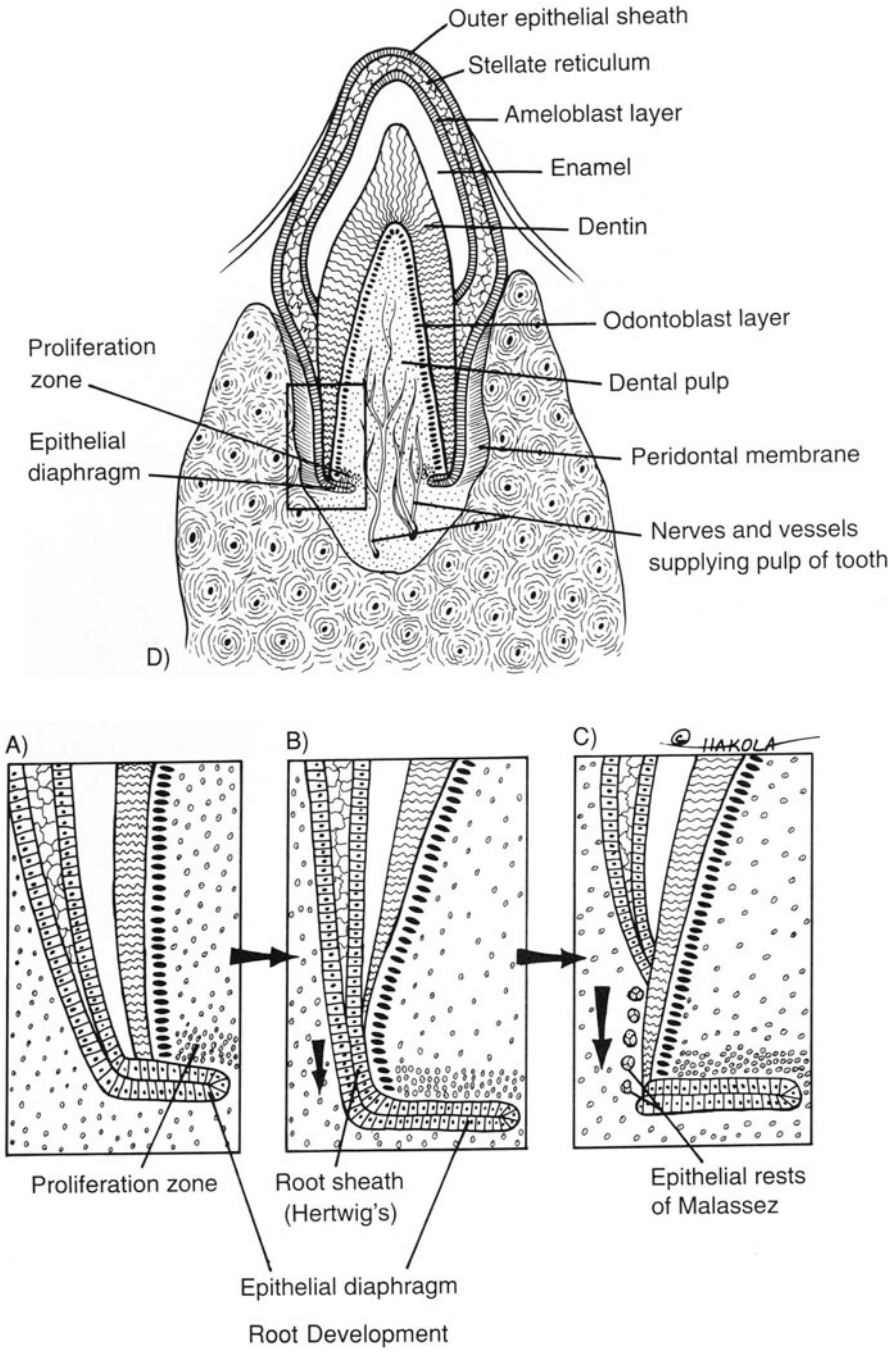


Figure 4.8. Root development (A) Epithelial diaphragm forms from inner and outer enamel epithelium at level of future cemented enamel junction. (B) Epithelial diaphragm remains relatively fixed with the crown rising by proliferation of coronal epithelial cells of the root sheath. (C) Entrapped epithelial remnants of root sheath within developing periodontal ligament (rests of Malassez). (D) Crown development completed prior to formation of root and periodontal structures.

formation and mineralization take place as in the tooth crown.

The root sheath forms the epithelial diaphragm prior to root formation. The double epithelial layer bends at the future cementoenamel junction, narrowing the opening of the developing tooth. The diaphragm remains relatively fixed, and proliferation of the coronal epithelial cells of the sheath raise the crown of the tooth (Fig. 4.8B). Epithelial cell proliferation slows relative to pulpal connective tissue as full root length is achieved. The apical opening is further narrowed by apposition of dentin and cementum. Differential growth of the epithelial diaphragm causes the formation of multiple roots.

Periodontal Development

As Hertwig's root sheath is lost, mesenchymal cells from the dental follicle come into contact with the outer dentin surface of the root and form the periodontal ligament and cementoblasts. Epithelial cells entrapped within the newly formed periodontal ligament are called rests of Malassez (Fig. 4.8C). Cementoblasts line the root surfaces and deposit cementum matrix on the dentin of the root. Mineralization follows once some matrix formation has occurred.

Table 4.4. Dental eruption ages and patterns.

	Average Age of Eruption	Average Age Shed
A. Primary		
1. Upper		
a. Central incisor	8–12 months	6–7 years
b. Lateral incisor	9–13	7–8
c. Cuspid (canine)	16–22	10–12
d. First molar	13–19	9–11
e. Second molar	25–33	10–12
2. Lower		
a. Central incisor	6–10 months	6–7 years
b. Lateral incisor	10–16	7–8
c. Cuspid	17–23	9–12
d. First molar	14–18	9–11
e. Second molar	23–31	10–12
B. Permanent		
1. Upper		
a. Central incisor	7–8 years	
b. Lateral incisor	8–9	
c. Canine	11–12	
d. First premolar	10–11	
e. Second premolar	10–12	
f. First molar	6–7	
g. Second molar	12–13	
h. Third molar	11–21	
2. Lower		
a. Central incisor	6–7 years	
b. Lateral incisor	7–8	
c. Canine	9–11	
d. First premolar	10–12	
e. Second premolar	11–12	
f. First molar	6–7	
g. Second molar	11–13	
h. Third molar	17–21	

As the tooth erupts (Fig. 4.8D, Table 4.4), the fibers of the periodontal ligament assume a functional alignment, and alveolar bone develops. Osteogenic cells in the dental sac differentiate into osteoblasts that produce osteoid (collagen and ground substance). Mineralization by hydroxyapatite crystals takes place between and within fibrils of the osteoid and is essentially uniform throughout the jaw. The radiodensity of alveolar bone seen around tooth roots is the result of tangential uptake of x-rays rather than of increased mineralization.

The dentogingival junction is formed by fusion of the basal cells of the oral mucosa with the outer enamel as the tooth erupts. This fusion results in continuity of the oral mucosa by transformation of the reduced enamel epithelium into squamous epithelium at the level of the cementoenamel junction. Toothbrush abrasion, periodontal disease, and tooth extrusion may violate the junction and expose the very sensitive cementin.

Developmental Abnormalities

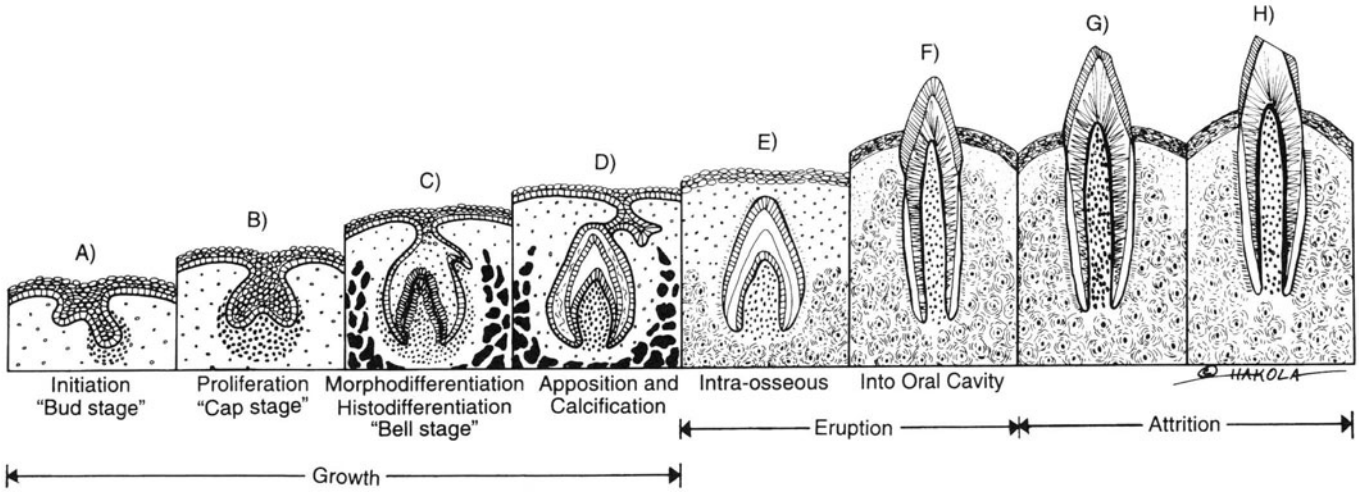
Disturbances in tooth development are not unusual. Enamel pearls primarily located in molar furcation areas result from the development of cells of Hertwig's root epithelium into ameloblasts that form enamel. Disintegration of the dental lamina from the pressure of cell proliferation and mesenchymal invasion may be incomplete, with residual cells persisting as epithelial pearls (Serres' pearls), which may develop into cysts or odontogenic tumors. Other growth disturbances can lead to congenitally missing primary or permanent teeth (anodontia), an excessive number of teeth (supernumerary teeth), abnormally shaped teeth, and fusion of adjoining developing dental elements. The etiologies of these anomalies are not well understood. Nutritional, chemical, and infectious factors may harm the ameloblasts and cause enamel hypoplasia; genetic transmission (autosomal dominant trait) and cause *amelogenesis* or *dentinogenesis imperfecta*.

All stages of tooth development are present at birth (Table 4.5). The cusp tip of the first permanent molar is the only tooth of the permanent set radiographically observable at birth. The formation and mineralization of teeth, particularly of the permanent dentition, remain vulnerable to chemical or traumatic insult during a child's early years.

Shedding of Primary Teeth

The physiologic phenomenon of primary teeth resorption allows the normal eruption of the permanent dentition. The bone between the primary tooth and its successor is resorbed prior to or simultaneous with the resorption of the primary tooth. Resorption begins on the lingual aspect of the root surface of the anterior tooth because of the location of the developing tooth;

Table 4.5. Schematic illustration showing the life cycle of a tooth (Modified from Orban).



primary molar resorption begins at the apices of the teeth because the developing permanent tooth bud is located beneath or between the roots. Formation of primary tooth roots is usually completed by 3 to 4 years of age. Hard tissue resorption by odontoclasts may begin at any time and is not necessarily a continuous process. Phagocytosis is probably the mechanism for pulp removal. Although reparative phases may take place during the resorption process, repair ultimately gives way to exfoliation of the primary tooth.

Shedding of the primary teeth is thought to result from pressure stimulation from the developing permanent tooth. A secondary cause may be the forces exerted on the primary tooth periodontal ligament by mastication. In the absence of permanent successors, primary teeth can be retained indefinitely if protected from masticatory forces. Internal resorption is an unimportant factor in the shedding of primary teeth.

Ankylosis of primary molars occurs in 8% to 10% of children. Although the affected teeth initially appear to have a normal eruption pattern, a bony union, the mechanism of which is not understood, develops between the alveolar bone and dentin or cementum of the primary teeth. Clinically, it appears that the crown of the affected tooth falls below the normal occlusal level, sparing the tooth from masticatory forces. Although most primary teeth eventually exfoliate, developmental abnormalities of the permanent dentition may include a misdirected eruption path, disturbances of eruption patterns and timing, and dental arch crowding with subsequent difficulty with extraction.

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CHAPTER 5

Oral Anatomy

James W. Ferraro

Oral Anatomy

The oral cavity is the first part of the digestive tract. It has many functions, such as the port of entry and the mastication of food. It also contains the organs of taste and salivary production, which not only lubricates food to facilitate swallowing, but also contains enzymes that initiate digestion.

The oral cavity is lined throughout by a mucous membrane, which is stratified squamous epithelium. These mucous membranes vary in different areas of the oral cavity in accordance with the functions and mechanical influences that bear upon them. Around the teeth and on the hard palate, for example, the mucous membrane is exposed to mechanical influences in the mastication of rough and hard food and, as a result, it is nonresiliant and tightly attached to the underlying bone. Those areas on the cheeks and floor of the mouth that are protected by the tongue do not have as much friction associated with them, and are therefore loosely attached to the underlying periosteum.

The mucous membrane is composed of two layers: the surface epithelium and the underlying lamina propria. It is attached to the underlying structures by a layer of connective tissue, the submucosa, which varies in character in different areas of the oral cavity. A basement membrane separates the lamina propria from the stratified squamous epithelium. The epithelium consists of several layers of cells that flatten out as they approach the surface and are connected to each other by intercellular bridges (see Fig. 5.1).

The innermost of these layers is the basal layer, consisting of cuboid cells that affect the attachment of the epithelium to the basement membrane of the connective tissue by numerous short basal processes that fit into grooves of the lamina propria. As we progress up from the basal layers, the next layers are the so-called "prickle cells," which are several layers of polyhedral cells with intracellular bridges giving them a spinous or prickly appearance. The basal and prickle-cell layers together are sometimes referred to as the stratum germinativum. Regeneration of epithelial cells, also at the surface, occurs by mitotic division of cells in the deepest layer or basilar areas (Fig. 5.1).

The lamina propria is a dense connective tissue layer of variable thickness. Its papillae, which indent the epithelium, carry both blood vessels and nerves, some of which actually pass into the epithelium. The arrangement of the papillae increases the area of contact between the lamina propria and the epithelium and facilitates the exchange of nutrients between blood vessels and epithelium.

The submucosa is the next layer and consists of connective tissue of varying thickness and density. It attaches the mucous membrane to the underlying structures of periosteum and bone (Fig. 5.1). Glands, blood vessels, nerves, and also adipose tissue are present in this layer. It is in the submucosa that the larger arteries and veins divide into smaller branches that enter the lamina propria. Here they again divide to form a subepithelial capillary network in the papillae. The blood vessels are accompanied by a rich network of lymph vessels, which play an important part in the drainage of the mucous membranes. The sensory nerves of the mucous membrane traverse the submucosa. These nerve fibers are myelinated but lose their myelin sheath in the mucous membrane before splitting into their end arborizations. The blood vessels of this layer are accompanied by nonmyelinated visceral nerve fibers that supply their smooth muscles; other visceral fibers supply the glands.

The oral cavity can be divided into two parts: the vestibule consisting of the lips and cheeks and the oral cavity proper, consisting of the teeth and alveolar ridges.

The lips can be divided into four parts: the skin on the outside, vermilion border, the transitional zone, and the true mucous membrane.

The skin is covered by a cornified epithelium of moderate thickness with a few short papillae in the lamina

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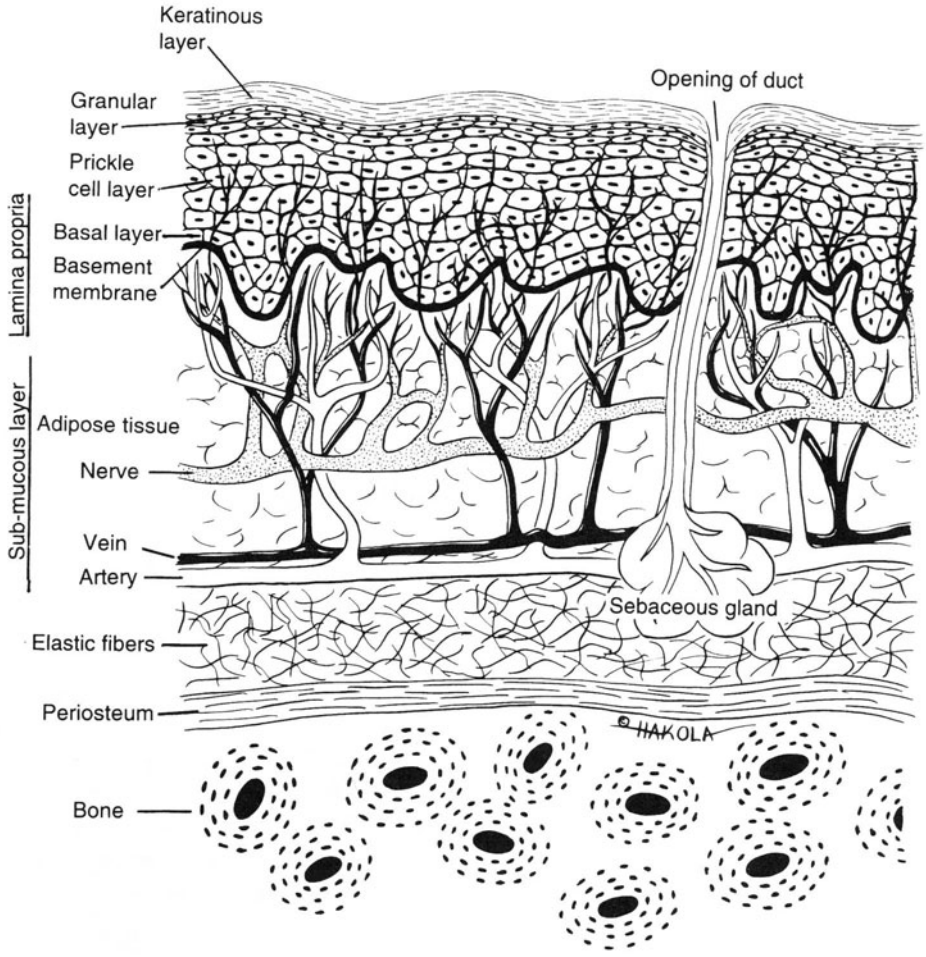


Figure 5.1. Schematic illustration of the oral mucosa.

propria. It also has hairs, sebaceous glands, and sweat glands within it. The vermilion border separates the cornified skin with the noncornified mucous membrane and is present only in humans. The mucosa of the lips is divided into the transitional zone and the true mucosa. The transitional zone is characterized by numerous, densely arranged, long papillae in the lamina propria, carrying with them large loops of capillaries that run close to the surface. (This is why the vermilion is red in color.) This transitional zone contains only a few sebaceous glands and is therefore subject to drying if not moistened repeatedly by the tongue.

Subdivisions of the Oral Mucosa

In studying any mucous membrane the following features should be considered: (1) the type of covering epithelium; (2) the structure of the lamina propria, especially as to its density, thickness, and presence or lack of elasticity; and (3) its fixation to the underlying structures such as the submucous layer. A submucosa may be present or absent as a separate and well-defined layer. The looseness or density of this layer determines whether the mucous membrane is moveable or immovably attached to the deeper layers.

The true mucous membrane of the lips and cheeks is characterized by a relatively thin, noncornified epithelium with a thin lamina propria. In these areas, where the lining mucosa covers muscles (lips, cheeks, and undersurface of the tongue) the submucosa is fixed to the underlying fascia of the muscles. The mucosa here is smooth and very elastic, and acts as a safeguard in functional mastication. If it were not elastic it would fold and produce creases that could be traumatized when chewing if it protruded between the teeth. The mucosa of the soft palate is a transition zone between the mucosa of the lips and cheeks and that of the vestibule and floor of the mouth. The vestibule mucosa and that of the floor of the mouth are loosely attached and allow for the movement of these structures. The mucosa of the vestibule (alveolar mucosa) is quite pink and red in color and very flexible when compared to the attached mucosa over the gingiva.

The attached mucosa is specialized depending on its position within the oral cavity. It is subjected to the forces and friction of mastication (called masticatory mu-

cosa), and thus is thick and cornified with a dense, firm lamina propria. It is tightly adherent to the teeth and bone because it is made of dense bands of fibrous connecting tissue that join the mucous membrane directly to the underlying periosteum and bone. The submucous space in the mucosa of the hard palate is subdivided into irregular and intercommunicating compartments of various sizes that are filled with fat in the anterior palate and glands in the posterior palate. The submucous space of the gingiva does not exist, and instead the lamina propria continues into the depth of the tissues and fuses directly with the periosteum of the alveolar process or the cervical region of the teeth.

Gingiva

The mucous membrane surrounding the teeth, the gingiva, is subjected to forces of friction and pressure in the process of mastication. The character of this tissue shows that it is adapted to meet these stresses. The gingiva is sharply limited on its outer surface of both jaws by a scalloped line (mucogingival junction), which separates it from the alveolar mucosa. The gingiva is normally pink, sometimes with a grayish tinge, a variable partly caused by differences in the thickness of the stratum corneum. The alveolar mucosa, on the other hand, is red, showing numerous small vessels close to the surface. A similar line of demarcation is found on the inner surface of the lower jaw between the gingiva and the mucosa on the floor of the mouth. In the palate, there is no sharp dividing line because of the dense structure and firm attachment of the entire palatal mucosa.

Normally, the epithelium of the gingiva is cornifed on its surface and contains a granular layer. In the absence of cornification there is no granular layer and the flat surface cells contain nuclei that are frequently pyknotic.

The epithelium that covers the margin of the gingiva also continues into the epithelial lining of the gingival sulcus. The cells of the basal layer here may contain pigment granules (melanin). While pigmentation is a normal occurrence in blacks it may also be found in Caucasians, especially those with a dark complexion. When found, it is most abundant in the basal areas of the interdental papillae. If, however, there is increased pigmentation of the interdental papillae, especially when associated with increase in pigmentation of the skin, Addison's disease can be suspected.

The lamina propria of the gingiva consists of dense connective tissues that are not highly vascular. The papillae of the attached gingiva are characteristically long, slender, and numerous. The presence of these numerous papillae permits a sharp demarcation of the gingiva with that of the alveolar mucosa, which usually have fewer and flatter papillae. The tissues of the lamina propria contain only a few elastic fibers that are, for the most part, confined to the walls of the blood vessels. The gingival fibers of the periodontal membrane enter into the lamina propria, attaching the gingiva firmly to the cementum. The gingiva is immovable and firmly attached to the periosteum of the alveolar bone because of the presence of large, coarse collagen bundles that extend from the lamina propria into the bone.

The gingiva may be divided into the free gingiva and attached gingiva. The dividing line between these two parts of the gingiva is the free gingival groove, which runs parallel to the margin of the gingiva at a distance of 0.5 to 1.5 mm (Fig. 5.2). The free gingival groove is, on histologic section, a shallow, v-shaped groove corre-

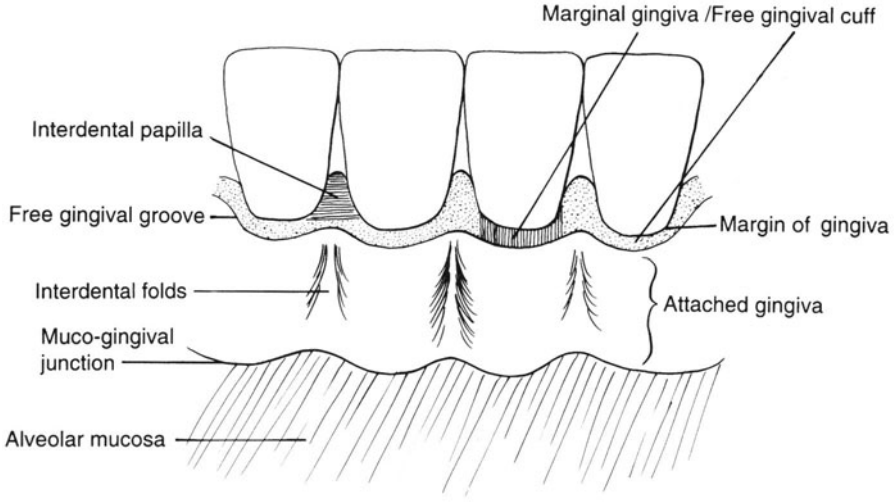


Figure 5.2. Diagram illustrating the surface characteristics of the gingiva of the lower anterior teeth.

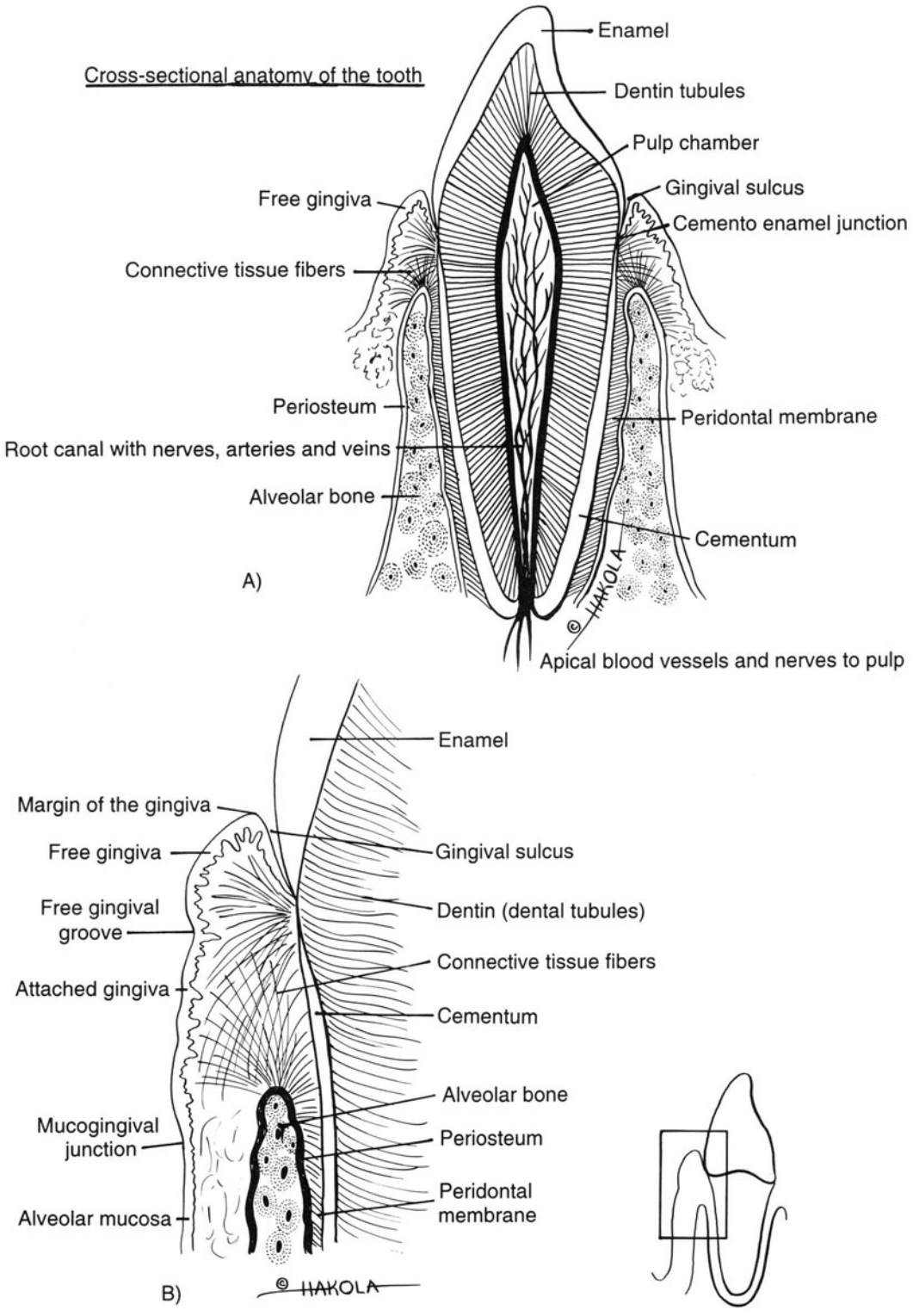


Figure 5.3. Diagram illustrating the anatomy of the tooth and the gingival sulcus.

sponding to the heavy epithelial ridge that divides the free and attached gingiva (Fig. 5.3). The free gingival groove develops at the level of, or somewhat apical to, the bottom of the gingival sulcus. In some cases, the free gingival groove is not as well defined as in others, and then the division between the free and the attached gingiva is not as clear.

The attached gingiva is characterized by high connective tissue papillae elevating the epithelium the surface of which appears striped. The stippling is most probably an expression of functional adaptation to mechanical impacts. The degree of stippling varies with different individuals. The disappearance of stippling is an indication of edema, secondary to chronic inflammation such as gingivitis. It is important to distinguish between attached gingiva and alveolar mucosa. This is because teeth will not erupt through alveolar mucosa but will most assuredly erupt through attached gingiva. This is especially important in patients in which bone grafting is being performed in the region of the alveolar clefts. If the erupting cuspid is covered by attached gingiva it will not properly erupt into the oral cavity, whereas if attached gingiva is brought over the area of the bone graft the erupting cuspid can easily erupt into the oral cavity in its normal dental position. It is also important not to bring alveolar mucosa down around the necks of erupted teeth because the alveolar mucosa will not attach to the adjacent tooth structure with a normal architectural pattern. Periodontal pockets can easily develop, the mucosa appears red and beefy, and it will not withstand the forces and friction of mastication as attached gingiva will.

Attached gingiva appears slightly depressed between each tooth, corresponding to the depression of the alveolar bone process between eminences of the sockets. In these depressions, the attached gingiva often form slight vertical folds (Fig. 5.2).

The interdental papilla is that part of the gingiva that fills the space between two adjoining teeth and is limited at its base by a line connecting the margin of the gingiva at the center of one tooth to the center of the other. The interdental papilla is composed of free gingiva and attached gingiva in various relations, depending largely upon the relationship of the neighboring teeth.

Hard Palate

As we have said before, the mucous membrane of the hard palate is tightly fixed to the underlying bone and, therefore, immovable. The color is pink, like that of the gingiva. The epithelium is uniform in character throughout the hard palate with a rather thick cornified layer and numerous long pegs. The lamina propria is thicker in the anterior than in the posterior parts of the palate. There are zones in the palate that must be distinguished: (1) the gingival region, adjacent to the teeth; (2) the palatine raphe, extending from the incisive palatine papilla posteriorly; (3) the anteriorlateral area, or fatty zone between raphe and gingiva; and (4) the posterolateral zone or glandular zone, between raphe and gingiva.

The gingival region shows the same structure as the other regions of the gingiva. Therefore, in this zone, a submucous layer cannot be differentiated from the lamina propria or periosteum. Similarly, the layers of the lamina propria, submucosa, and periosteum cannot be distinguished in the palatine raphe or median area. If the patient presents with a palatine torus, the mucous membrane over the torus is noticeably thin and the otherwise narrow raphe spreads over the entire torus or is absent altogether.

In the lateral areas of the hard palate, in both the fatty and glandular zones, the lamina propria is fixed at the periosteum by strands of dense fibrous connective tissue, which is at right angles to the surface and divides the submucous layer into irregularly shaped spaces. The distance between lamina propria and periosteum is similar in the anterior as it is in the posterior parts of the palate. As we mentioned earlier, the anterior zone in the connective tissue spaces contains the fat, while in the posterior part, lobules of mucous glands are packed into the spaces. The glandular layer of the hard palate extends posteriorly to the soft palate.

In the sulcus between the alveolar process and the hard palate, the palatine vessels and nerves are found surrounded by loose connective tissue. This area, being wedge-shaped in cross-section, is relatively large in the posterior parts of the palate and gradually diminishes in size as we move anteriorly (Fig. 5.4).

The pear-shaped or oval incisive papilla is formed of dense connective tissue. It lies at the junction between the premaxilla and maxilla and contains the oral part of the vestigial nasopalatine ducts, also known as Stenson's organ. These are blind epithelial ducts of varying lengths and are lined by a simple columnar epithelium, rich in goblet cells, with small mucous glands opening into the lumen. Frequently the ducts are bordered by small irregular islands of hyalin cartilage, the remnants of the capsule of Stenson's organ. Stenson's organ is present in most mammals and is considered an auxiliary olfactory sense organ. The cartilage is sometimes found in the anterior parts of the papilla; it then shows no apparent relationship to the nasopalatine ducts.

In the region of the palatine papilla, epithelial pearls may be found within the lamina propria. They consist of concentrically arranged epithelial cells, which are frequently cornified. They have remnants of the epithelium in the line of fusion between the palatine processes. Occasionally these may result in formation of a cystic

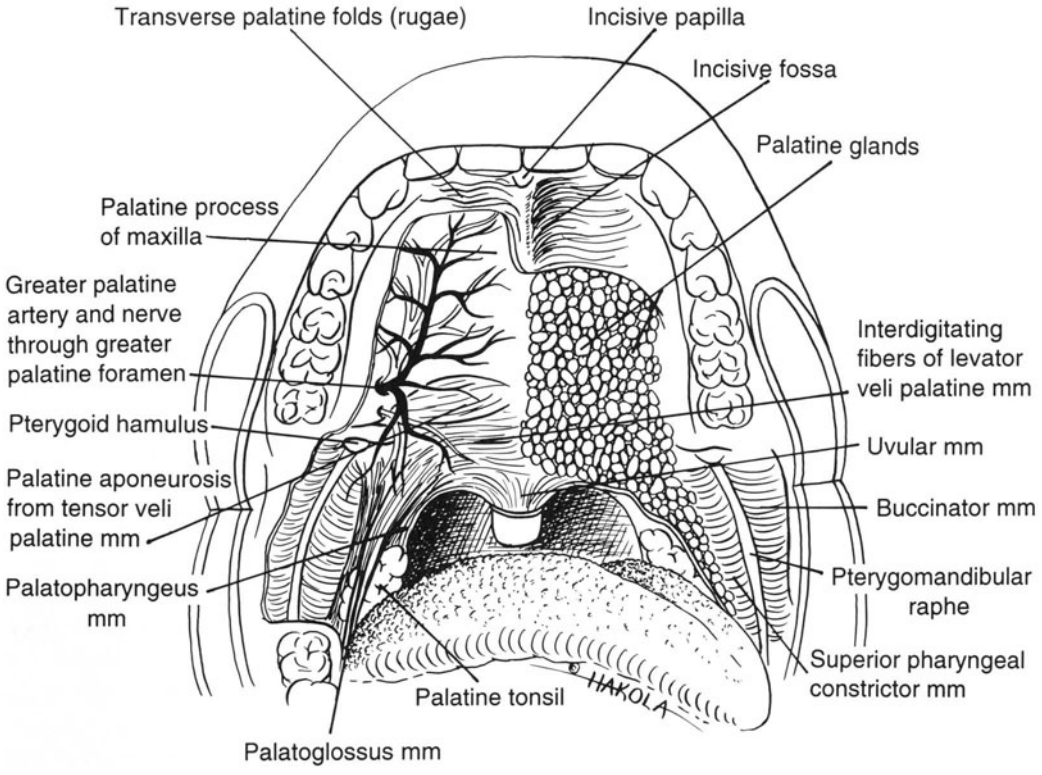


Figure 5.4. Cross-sectional anatomy of the palatal mucosa.

structure, known as a nasopalatine cyst. It occurs between the roots of the central incisor and when it expands it can cause separation of these roots away from the midline. The transverse palatine ridges (palatine rugae) are irregular and often asymmetric in humans. There are ridges of mucous membrane extending laterally from the incisive papilla with a core of dense connective tissue with finely interwoven fibers (Fig. 5.4).

Vestibular Fornix and Alveolar Mucosa

The vestibular fornix is the area where the mucosa of lips and cheeks reflects to become the mucosa covering the jaws. The mucous membrane of the cheeks and lips is firmly attached to the buccinator muscle in the cheeks and the orbicularis oris muscle in the lips. In the fornix, the alveolar mucosa is loosely connected to the underlying structures and thus permits the necessary movements of lips and cheeks. The mucous membrane covering the outer surface of the alveolar process is loosely attached to the periosteum in the area close to the fornix. It continues into, but is sharply limited from, the gingiva, which is firmly attached to the periosteum of the alveolar crest and to the teeth.

Gingival and alveolar mucosae are separated by a scalloped line, muco-gingival junction (Fig. 5.2). The altered appearance of tissues on either side of this line (gingival tissue being pink, alveolar mucosae being red) is due to a difference in their structures and vascularity. The attached gingiva is stippled, firm, thick, lacks a separate submucous layer, is immovably attached to the bone, and has no glands. The gingival epithelium is thick and cornified; the epithelial ridges and the papillae of the lamina propria are high. The alveolar mucosa is thin and loosely attached to the periosteum by a well-defined submucous layer of loose connective tissue and may contain small mucous glands. The epithelium is thin, not cornified, and the epithelial ridges and papillae are low and often entirely missing. The mucous membrane on the floor of the oral cavity is thin and loosely attached to the underlying structures to allow for the

free mobility of the tongue. The epithelium is not cornified and the papillae of the lamina propria are short. The submucosa contains adipose tissue. The sublingual glands lie close to the covering mucosa. The sublingual mucosa joins the lingual gingiva in a sharp line that corresponds to the mucogingival line on the vestibular surface of both jaws. At the inner border of the horseshoe-shaped sublingual sulcus, the sublingual mucosa reflects onto the lower surface of the tongue and continues as the ventral lingual mucosa.

The mucous membrane of the inferior surface of the tongue is smooth and relatively thin. The epithelium is not cornified; the papillae of the connective tissue are numerous but short. Here, the submucosa cannot be identified as a separate layer; it binds the mucous membrane tightly to the connective tissue surrounding the bundles of the striated muscles of the tongue.

Soft Palate

The mucous membrane on the oral surface of the soft palate is highly vascularized and of reddish color, noticeably differing from the pale color of the hard palate. As with the alveolar mucosae, the papillae of the connective tissue are few. The lamina propria shows a distinct layer of elastic fibers separating it from the submucosa. The latter is relatively loose and contains an almost continuous layer of mucous glands. Typical oral mucosa continues around the free border of the velum palatinum and is replaced, at a variable distance, by nasal mucosa with a pseudostratified, ciliated, columnar epithelium.

The Tongue

The superior surface of the tongue is rough and irregular. A v-shaped line divides it into an anterior part, or body, and a posterior part, or base of the tongue. The former comprises about two thirds of the length of the organ, the latter forming the posterior one third. The fact that these two parts develop from different areas of the branchial region accounts for the different source of nerves of general sense: the anterior two thirds is supplied by the trigeminal nerve through its lingual branch with taste supplied by the chordae tympani, a branch of the seventh cranial nerve. The posterior one third of the tongue is supplied by the glossopharyngeal nerve.

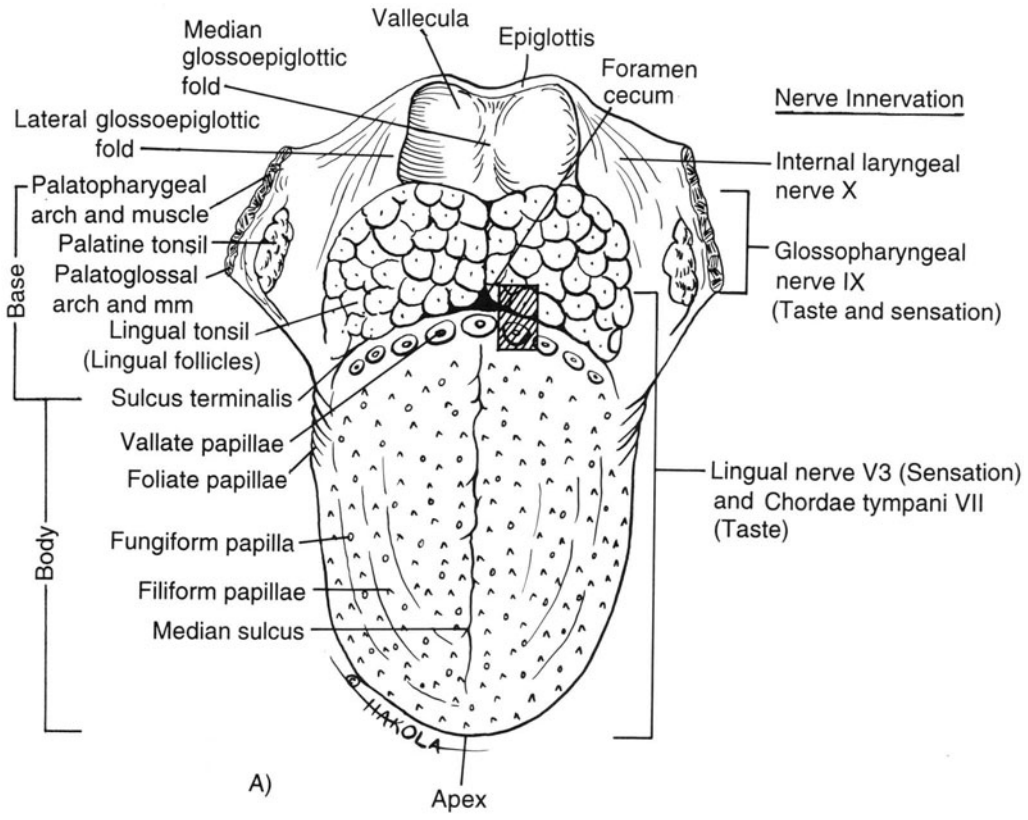
The body and base of the tongue differ widely in the structure of their covering mucous membrane. On the anterior part are found numerous fine-pointed, cone-shaped papillae that give it a velvet-like appearance. These projections, the filiform papillae (thread-shaped) are built of a core of connective tissue that carries secondary papillae. The covering epithelium is cornified, especially at the apex of the papillae. This epithelium forms hairlike tufts over the secondary papillae of the connective tissue. The filiform papillae do not have any taste buds.

Interspersed between the filiform papillae are the isolated mushroom-shaped or fungiform papillae, which are round, reddish prominences. Their color is derived from a rich blood supply visible through the relatively thinner epithelium. Some fungiform papillae contain a few taste buds.

Along the dividing v-shaped line, between the body and base of the tongue, are found the vallate or circumvallate (walled-in papillae); they are 8 to 10 in number. They do not protrude above the surface of the tongue, but are bounded rather by a deep and circular furrow that seems to cut them out of the substance of the tongue. They are slightly narrower at their base. Their free surface shows numerous secondary papillae, which are covered by a thin and smooth epithelium. On the lateral surface of the vallate papillae and occasionally on the walls surrounding them, the epithelium contains numerous taste buds. Into the trough open the ducts of small albuminous glands (von Ebner's glands), which serve to wash out the furrows into which the soluble elements of food penetrate to stimulate the taste buds.

At the angle of the v-shaped line on the tongue is found the foramen cecum, which is a remnant of the thyroglossal duct. Posterior to the circumvallate papillae, the surface of the tongue is irregularly studded with round or oval prominences known as the lingual follicles. Each of the latter show one or more germinal centers. Most of these prominences have a small pit at the center, the lingual crypt, which is lined with stratified squamous epithelium. Innumerable lymphocytes migrate into the crypts through the epithelium. The ducts of the medium-sized posterior lingual mucous glands open into the crypts. Together the lingual follicles form the lingual tonsil (Fig. 5.5).

On the lateral border of the posterior parts of the tongue, sharp parallel furrows of varying length can be observed. They bound narrow folds of the mucous membrane and are the vestiges of the large foliate papillae found in many mammals. They also contain taste buds. The taste buds are small ovoid or barrel-shaped intraepithelial organs of about 80 microns thickness. They touch with their broader base the basement membrane, while their narrower tip almost reaches the surface of the epithelium. The tip is covered by a few flat epithelial cells, which surround a small opening, the taste pore. It leads into a narrow space between the peripheral ends of the sustentacular (supporting) cells of the taste bud. The outer supporting cells are arranged like the staves of a barrel; the inner and shorter ones are spindle-shaped. Between the latter are arranged 10 to 12



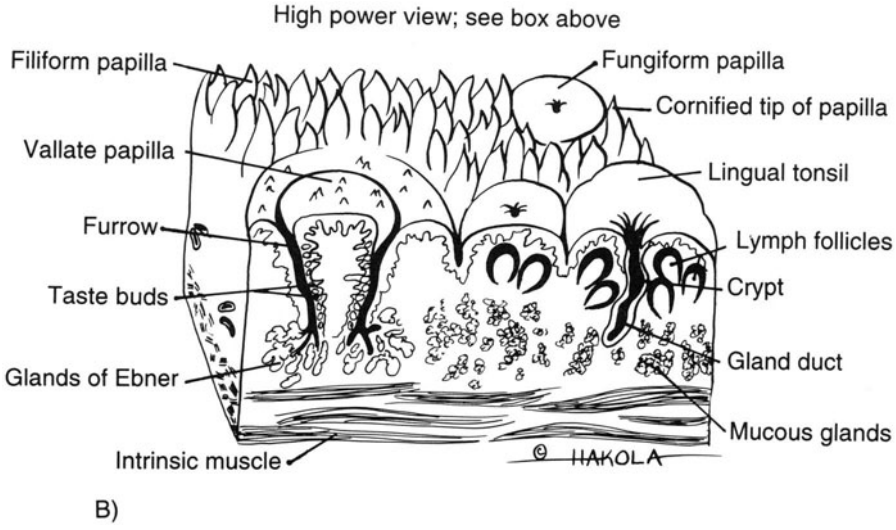


Figure 5.5. Topographical anatomy and innervation of the tongue.

neuroepithelial cells, which are the receptors of taste stimuli, they are thin, dark-staining cells that carry stiff hair-like processes at their superficial end. These hairs reach into the space beneath the taste pore.

A rich plexus of nerves is found below the taste buds. Some fibers enter the taste bud from the base and end in contact with the taste cells. Others end in the epithelium between the taste buds. Taste buds are numerous on the inner wall of the trough surrounding the vallate papillae, in the folds of the foliate papillae, on the posterior surface of the epiglottis and on some of the fungiform papillae at the tip and the lateral borders of the tongue.

The primary taste sensations, namely, sweet, salty, bitter, and sour, are not perceived in all regions of the tongue. Sweet is tasted at the tip, and salty at the lateral border of the tongue. Bitter and sour are recognized in the posterior part of the tongue, bitter in the middle, sour in the lateral areas. The distribution of the receptors for primary taste qualities can, diagrammatically, be correlated to the different types of papillae. They are mediated by different nerves. The circumvallate papillae recognize bitter taste, and the foliate papillae recognize sour taste. The taste buds on the fungiform papillae at the tip of the tongue are receptors for sweet taste, and those at the borders are receptors for salty taste.

Bitter and acid (sour) taste are mediated by the glossopharyngeal, and sweet and salty taste by the intermediofacial nerve via chorda tympani (Fig. 5.5).

Clinical Considerations

To understand the pathogenesis of periodontal diseases and the pathologic involvements of the different structures, it is essential to be thoroughly familiar with the structure of cementum, periodontal membrane, alveolar bone, and the structure of the marginal gingiva, gingival sulcus, and epithelial attachment, as well as their biologic relation to each other (Fig. 5.6). Periodontal dis-

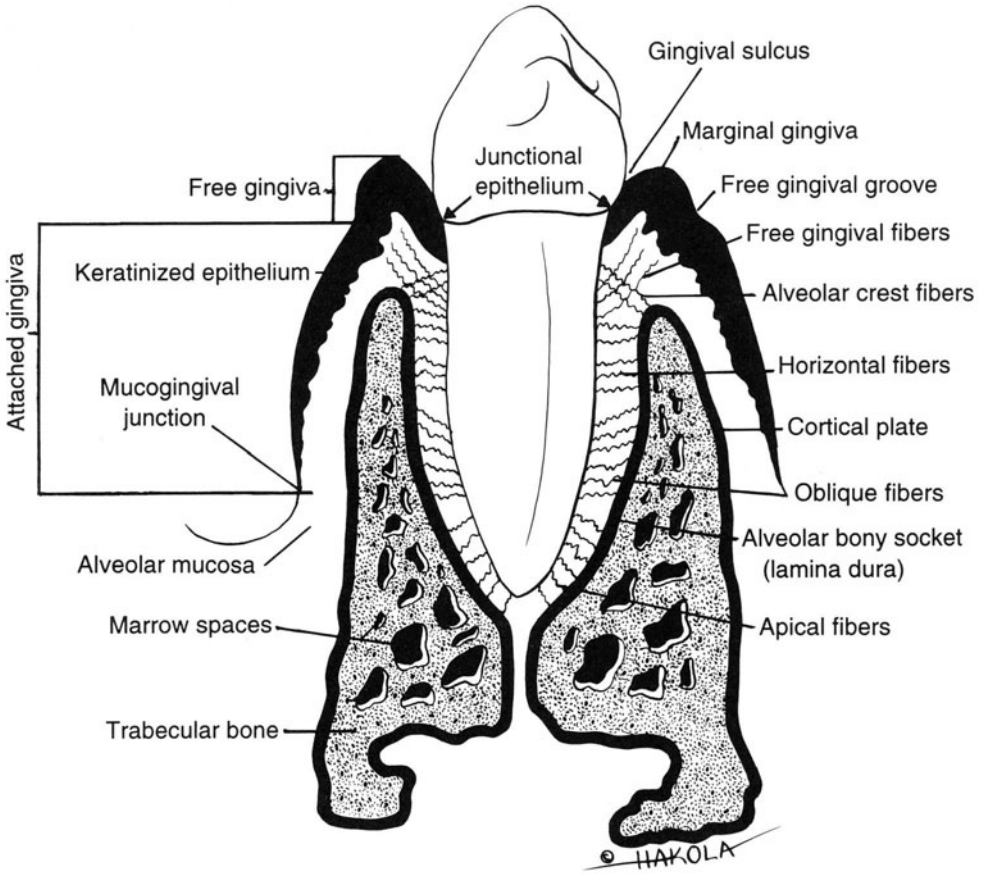


FIGURE 5.6. Cross-sectional anatomy of the gingiva, peridontal membrane, and the supporting bone.

turbances frequently have their origin in the gingival sulcus and marginal gingiva, leading to the formation of a deep gingival pocket. Moreover, the safe and speedy reduction of the depth of the gingival pocket is the primary objective of treatment. The superiority of any given method of treatment should be judged by its ability to accomplish this end whether the method be surgical, chemical, or electrical.

Dentition

In humans the teeth are a complicated organ composed of a core, the dental pulp, which is rich in nerves and blood vessels. The bulk of the tooth is formed from dentin, with that portion exposed to the oral cavity called the crown, which is covered with an ectodermal derivitive (enamel). The portion of the tooth embedded in the bony socket is covered by a mesodermal derivative (cementum) that is by its nature almost exactly like bone. The junction of the root and the crown is the cemento-enamel junction.

The color of a tooth is dependent on (1) whether it is deciduous or permanent, (2) enamel color and translucency, and (3) age of the tooth. The deciduous dentition is generally of a white color whereas the permanent dentition is more yellow. The enamel has a very faint yellow color whereas the dentin has a darker yellow color. In the areas of the crown where the enamel is plentiful, the translucency is less and therefore the tooth is whiter (occlusal or biting surface). That portion near the neck of the tooth has less enamel and therefore more of a yellow color. In older teeth where the enamel has been worn the yellow color is usually darker because the dentin has been exposed and shows its darker color. In older teeth the dentinal tubules become stained because of imbibition of the materials from our food and take on a more yellow hue. Also lower teeth are whiter than upper teeth, especially in the anterior dentition.

The size of the teeth varies so much among individuals that only mean values or ranges of various sizes can be given. It has been stated by Sicher and Tandler that the mesial distal diameter of a crown correlated somewhat with the body height. However, the total length of a tooth (especially its root length) has no relationship to the size of an individual (Table 5.1).

The size of the pulp chamber and the widths of the root canals vary according to age. In young developing teeth, the pulp chamber is wide as are the root canals, especially at the apex of the root because the root is incompletely developed (see Fig. 5.7A). As the development of the root continues and further dentin is laid down, the apex of the root narrows. It is also known that dentin formation continues throughout life (but at a very slow rate). This causes a continual narrowing of the pulpal space and the root canals. The narrowing is not the same in all portions of the pulp chamber. As attrition of the occlusal surface occurs, dentin is laid down in the occlusal portion of the pulp chamber as a protective mechanism for the pulp. However, this apposition of dentin is irregular because the attrition of the crown is irregular, and therefore many patterns of pulp chambers can be seen on x-rays (Fig. 5.7B).

Once the tooth is developed and it erupts through the gingival surface, a gingival margin and cuff forms at the junction of the dento-enamel junction. The gingival surface is not attached to the crown of the tooth, but rather is attached below the cemento-enamel junction by collagenous nonelastic fibers called Sharpey's fibers of the alveolar crest fibers of the periodontal membrane. It is these Sharpey's fibers that attach the bone to the cementum, acting as a "shock absorber" and thereby protecting the tooth and the adjacent bone from the constant trauma of chewing. These fibers make up the bulk of the periodontal membrane. The periodontal membrane or periodontium is attached from the junction of the crown and the root and extends all the way down around the apex of the fully developed root as well as around the gingiva at the cemento-enamel junction (see Fig. 5.6). It is divided into three segments:

1. The gingival segment where the free gingival fibers attach to the cementum near the neck of the tooth.
2. The transeptal or interdental ligament. These fibers in a strict sense are not contained in the periodontal space but run across the interdental space from one tooth to the next and serve to unite all the teeth of one arch into a functional unit.
3. The alveolar ligament, which is horizontal and oblique fibers running from the alveolar bone to the cementum of the root.

Table 5.1. Mean lengths of teeth.

	Deciduous	Permanent
Upper central incisor	18 mm	24 mm
Upper lateral incisor	16 mm	22.5 mm
Upper cuspid	20 mm	27 mm
Upper first bicuspid	—	21.7 mm
Upper second bicuspid	—	21.5 mm
Upper first molar	16 mm	21.3 mm
Upper second molar	18 mm	21.2 mm
Upper third molar	—	Too Variable
Lower central incisor	17 mm	21.4 mm
Lower lateral incisor	17 mm	23.2 mm
Lower cuspid	19 mm	25.4 mm
Lower first bicuspid	—	22.7 mm
Lower second bicuspid	—	23.2 mm
Lower first molar	15.5 mm	22.8 mm
Lower second molar	18 mm	22.8 mm
Lower third molar	—	Too Variable

Adapted from Dubrul: Sicher's Oral Anatomy, 7th ed, 1980, with permission by author.

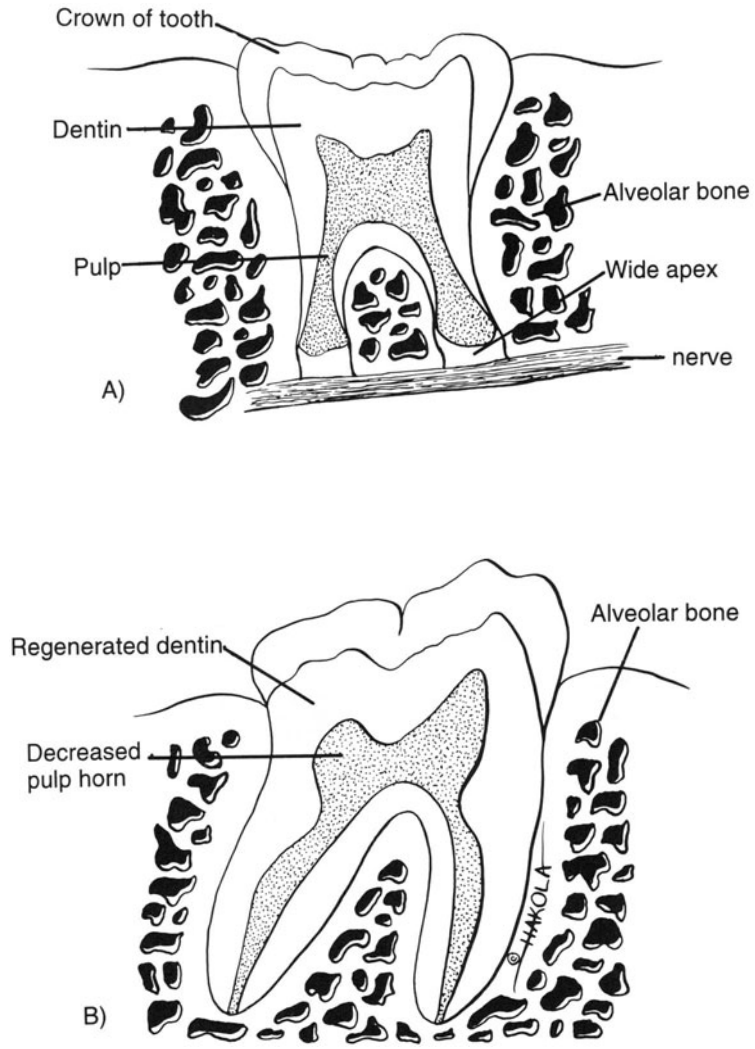


Figure 5.7. (A) Cross-sectional area of unerupted lower first molar of a child 4 1/2 years of age showing developing root with its wide apex. (B) Cross-sectional area of adult lower molar showing cuspid abrasion with protective regeneration of the dentin.

If there is any breakdown of the periodontium from inflammation (periodontitis) there will be a destruction or degeneration of the periodontal membrane (periodontosis). Associated with this is the inflammatory destruction of the adjacent alveolar bone. This results in progressive weakening of the tooth in the socket because of its lack of boney support. More teeth are lost because of periodontal and bone destruction than are ever lost because of decay.

In a multirooted tooth the same structures are present and the attachment of the tooth itself is exactly the same as a single-rooted tooth, except that there are more roots, meaning that there are more areas of attachment of that tooth.

Also in the periodontal membrane are remnants of the epithelilal root sheeth of the developing tooth (as mentioned earlier in this chapter) called the epithelial rests of Malassez. They are elliptical segments that can appear as round islands of epithelial cells near the root of a tooth especially at the apex. They are of no consequence except they may proliferate in association with inflammation and have been implicated as forming the epithelial lining of a radicular cyst at the apex of the tooth.

In Figure 5.8 a cross-section of a molar tooth is shown. The pulp canal in the roots and the main pulp chamber in the crown, with its arterial, venous, lymphatic, and neural structures, are seen as they pass from the pulp

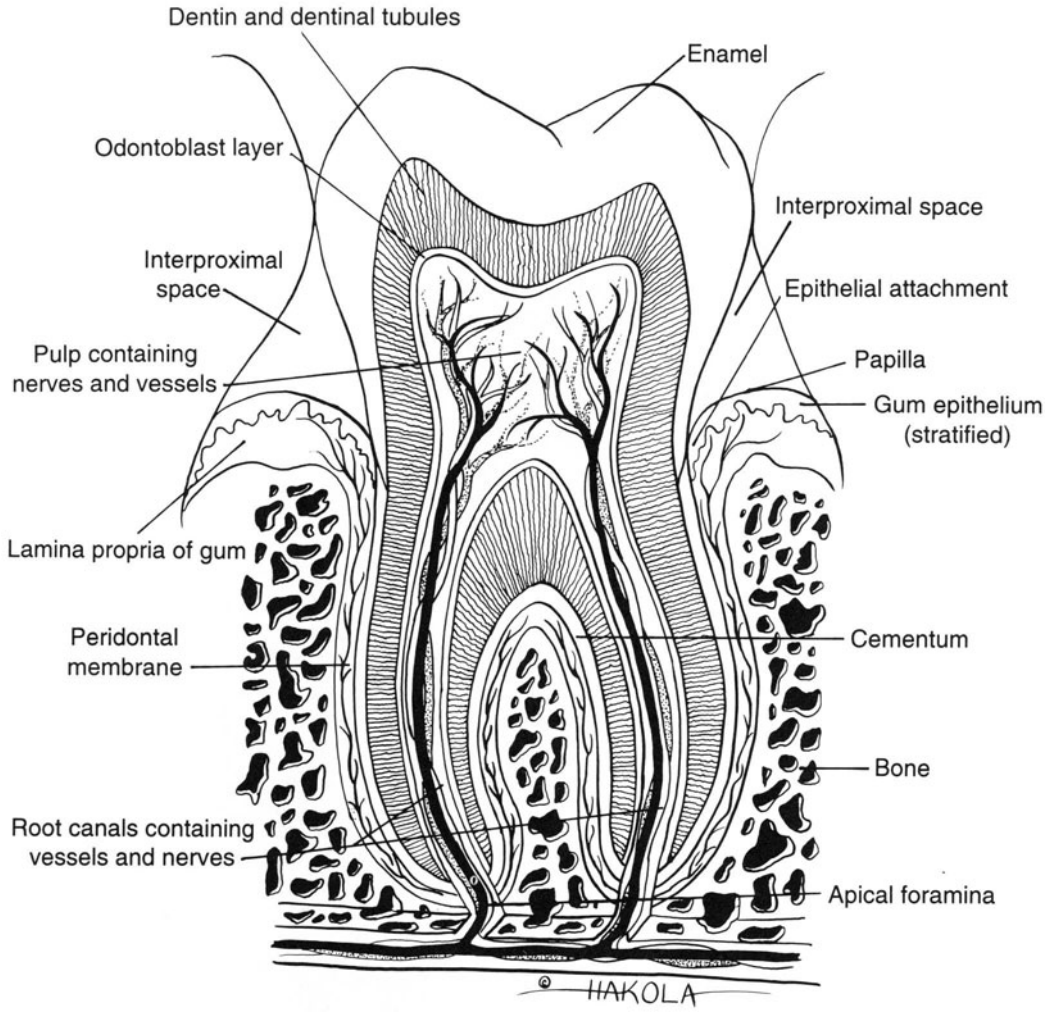


Figure 5.8. Cross-sectional anatomy of a lower molar.

chamber through the apex of the fully formed root. Nerves and vessels are plentiful as they exit through the apex of each tooth and have a wide communication with the blood supply coming from the adjacent bone through the periodontal membrane and the adjacent mucosa. If we consider the presence of a wide plexus we can easily understand how a tooth's pulp can be removed and yet the viability of the tooth structures can be maintained.

Sensibility and Viability

The term viable tooth tissue refers to a tooth with an intact blood supply either from the pulp or the adjacent tooth tissue. The term sensate refers to the ability of that tooth to emit the sensation of pain when the tooth's pulp is stimulated with an electric or cold stimulus. The two terms, sensate and viable, are not mutually exclusive. A tooth without a pulp (pulp chamber being removed by endodontics) still will be viable because of the blood supply coming in from the adjacent periodontal membrane, but that same tooth, however, will not emit a painful stimulus from a pulp testor and is therefore viable but nonsensate. The same principal occurs when we cut the maxilla in a Le Fort I osteotomy. All the teeth have lost their nerve supply because of the osteotomy and the transection of the posterior, middle, and anterior superior alveolar nerves. Yet if the blood supply is maintained via the palatal vessels the teeth will remain viable and be maintained in the alveolar process of the maxilla. If, however, the blood supply is cut and the segment becomes avascular and the viability is destroyed,

then the tooth and the adjacent bone will be sloughed from the arch. If the labial or palatal blood supply is maintained, segmental bony structures can be mobilized and moved into various positions.

Description and Nomenclature of the Dentition

Figure 5.9 shows the maxillary arch. If a line is drawn between the central incisors down the medial raphe of the maxilla, it divides the arch into right and left components. Dental terms such as mesial and distal are used to denote teeth position in the arch and surfaces on the tooth itself. For example, the central incisor is mesial to the lateral incisor, which is mesial to the cuspid (or canine). The first bicuspid is distal to the cuspid and the second molar is distal to the bicuspids and first molar. Similarly the central incisor has a mesial surface (toward the midline) and a distal surface (away from the midline). The lateral incisor and all the other teeth also have a mesial and distal surface depending on the surface and its relationship to the midline.

The lingual surface of all the teeth is that surface next to the tongue. The outer surface, however, is different. The anterior six teeth (central through cuspid on each side), because they are next to the lips, have a labial surface. The anterior teeth (bicuspids and molars), because they are next to the cheek, have a buccal surface.

The anterior six teeth have a sharp edge called the incisal surface, whereas the bicuspids and molars, because their surface is flatter and are used to grind food, have an occlusal surface. Thus, the surfaces of molars are mesial, distal, lingual, buccal, and occlusal. The surfaces of the anterior incisors are mesial, distal, labial, lingual, and incisal. The mandibular teeth surfaces are named exactly the same.

The anterior teeth have one more peculiar anatomical segment. Each of these teeth have a cingulum. The cingulum is actually an aborted cusp. In other words, if the cingulum "grew up" it would become a bicuspid, not an incisor or a canine. The cingulum is a small area, and is adjacent to the lingual surface of the anterior teeth.

The bicuspids erupt in the adult dentition where the deciduous molars are present in the primary dentition. Each of these teeth have two cusps, a buccal and a lingual cusp. These teeth are chewing and grinding teeth and can also tear food, but not as effectively as the incisors and canines. The molars are multicusped teeth (four or more) that also have multiple roots. Figure 5.10 shows the typical occlusal surface of the lower second molar with an anatomical description of the various cusps and grooves.

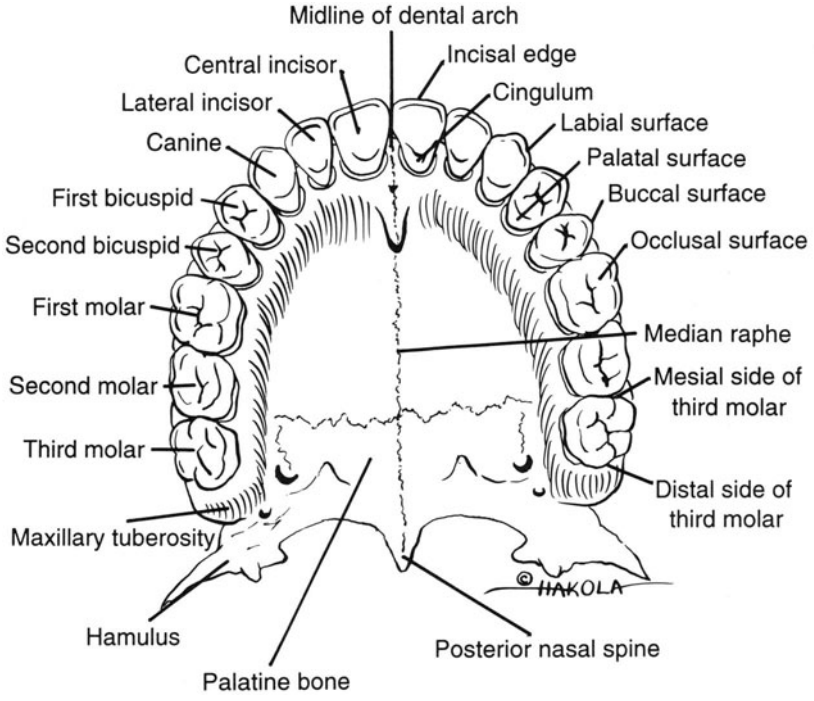


Figure 5.9. Anatomy of the palate and maxillary arch.

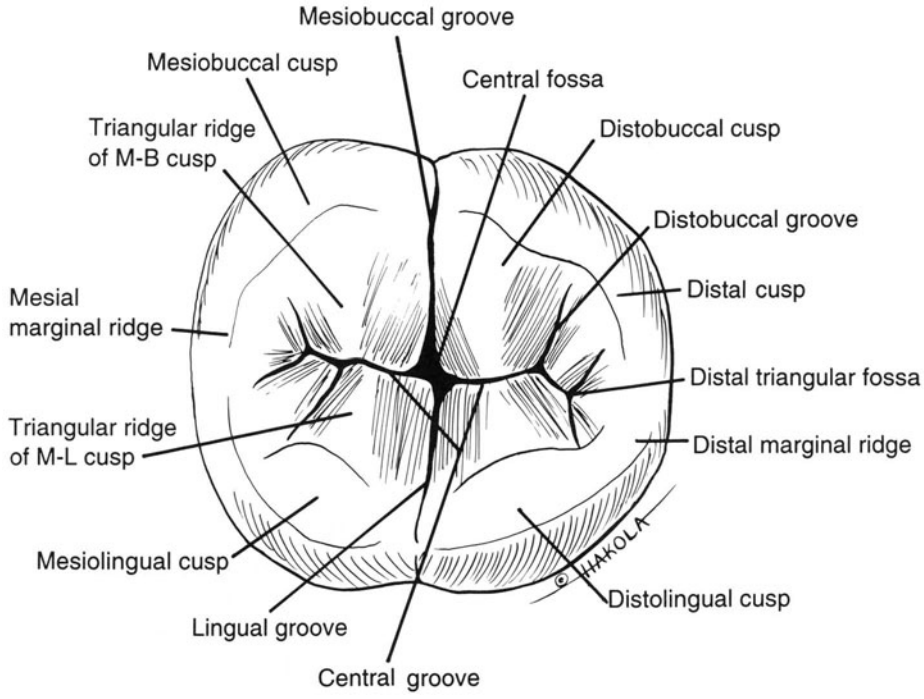


Figure 5.10. Anatomy of the occlusal surface of the lower second molar.

The shape of the dental arches varies considerably. However, in the average individual the arch has two curves in space. In the horizontal plane the arch form is somewhat u-shaped with the labial surfaces of the six anterior teeth. As we progress to the bicuspids this plane is continued. It is not until we reach the distal cusps of the first molar that the curve begins to bend toward the lingual (Fig. 5.11). This curve of the arch is important because it prevents the teeth from biting the cheek and gives clearance for the coronoid process of the mandible to move within the zygomatic arch.

The biting and chewing surfaces of the teeth in the arch also have a curve as you progress back toward the second molars. This curve is inclined slightly downward from the canine to the first molar and then rises again in the region of the molars. This curve is known as the Curve of Spee. For further discussion on the Curve or Spee, see "Occlusion" later in this text.

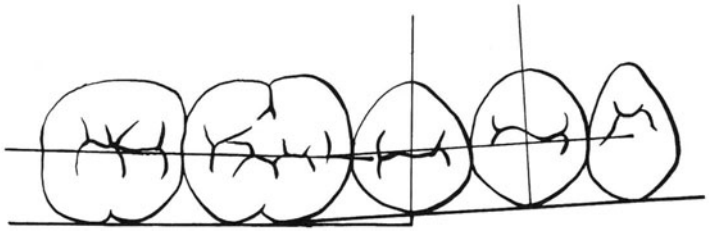


Figure 5.11. Buccal-lingual curve of the maxillary and mandibular posterior teeth.

Deciduous Dentition

The deciduous dentition closely resembles their permanent successors; however, they have smaller crowns and shorter root lengths. The anterior incisors have more curvature on their labial surface when compared to the adult version. The height of contour of the molars is closer to the gingiva than the permanent molars (see Fig. 5.12), thus making it much harder to get wires below the height of contour so that they will hold tight to the teeth when applying arch bars. As a result, acrylic splints, circumandibular and transmaxillary wires, drop wires from the puraform apertures, or zygomatic buttresses are frequently used to hold the arch bars in young patients.

The permanent dentition, in contrast, has contact points at the upper third of the clinical crown above the interproximal papillae. Fixation of arch bars in the permanent dentition can be easily secured with interdental wires if the dentition is intact.

The deciduous teeth in the anterior are replaced in kind by the permanent teeth. However, in the case of the deciduous molars they are replaced by permanent bicuspids. Because there are no bicuspids in the deciduous dentition there are only 20 teeth in the arch. In comparison, the 3 permanent molars erupt *de noveau* and as a result, there are 32 permanent teeth in the adult arch.

Table 5.2 compares the deciduous and permanent dentition as to nomenclature. Table 5.3 describes the deciduous and permanent eruption schedules.

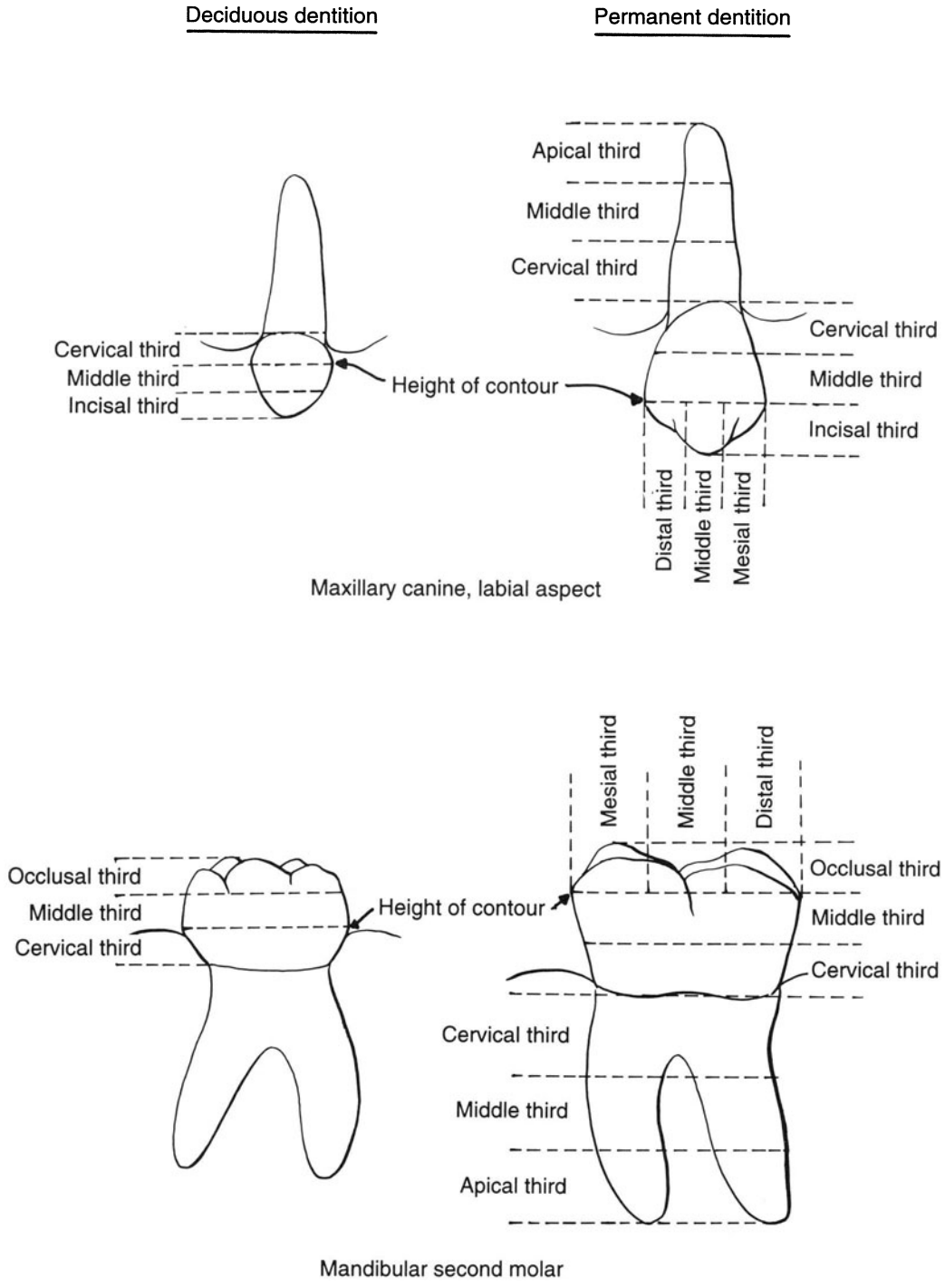
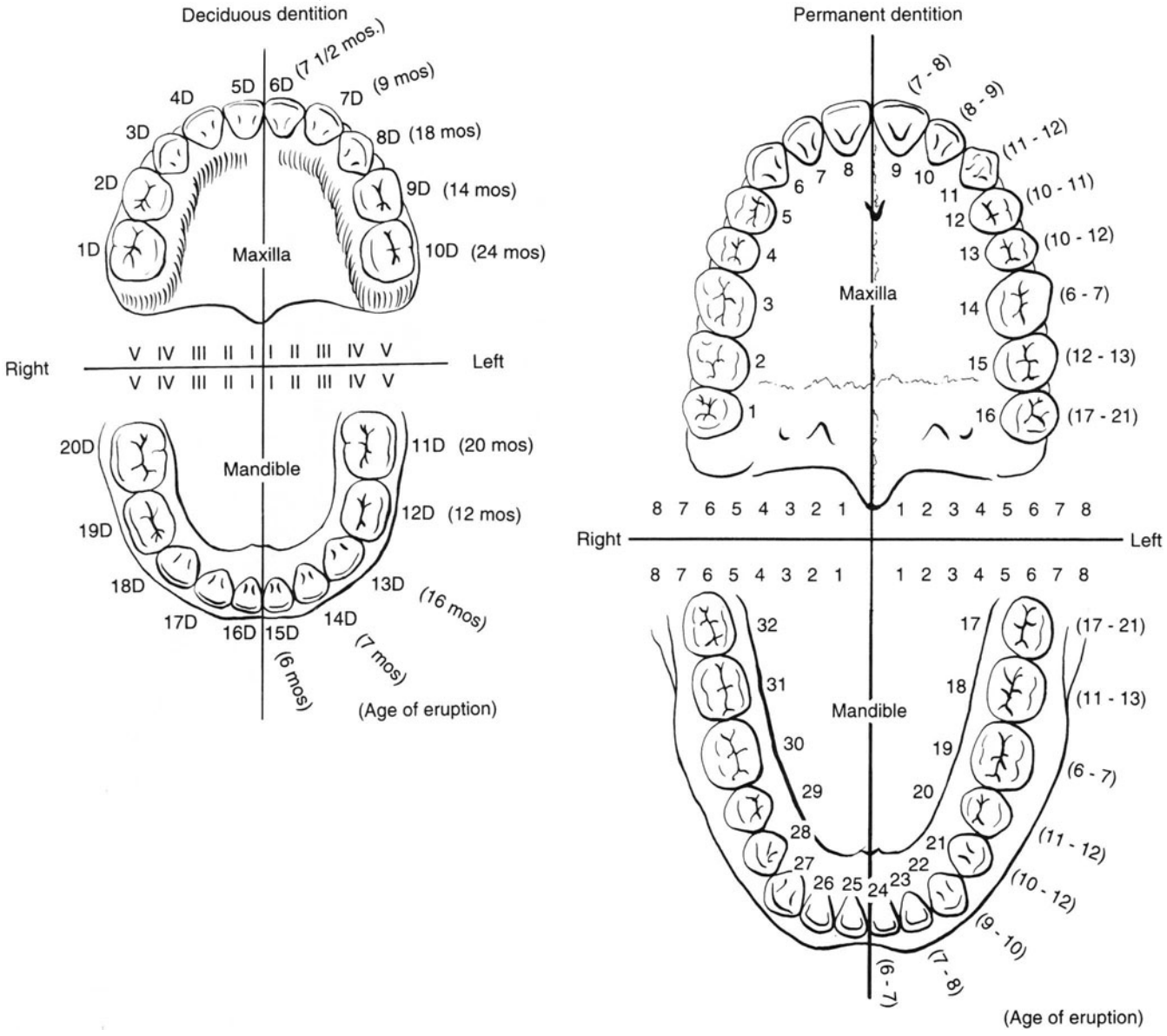


Figure 5.12. Anatomical comparison of the deciduous.

Table 5.2. Comparison of the nomenclature for deciduous and permanent dentition.



Dental Formulae

There are many formulas one can use to indicate the position of a permanent or deciduous tooth in the arch. In this text two basic methods are discussed. One method frequently used is where a cross was made and the vertical line would separate the right side from the left, while the horizontal line would separate the upper from the lower jaws. Looking at that cross it would be as though the patient were looking at you.

The deciduous dentition is marked in Roman numerals or letters (A through E) while Arabic numbers are used to represent any erupted permanent teeth. In Fig. 5.13 the left upper half of the maxillary primary dentition is seen. The deciduous central, lateral, cuspid, first, and second molars (I, II, III, IV, V) are present. Within the parenthesis, the partially erupted first permanent molars and the developing second permanent molar are noted. In Fig. 5.14 all permanent teeth in the maxilla are present and erupted. In this situation we are looking at

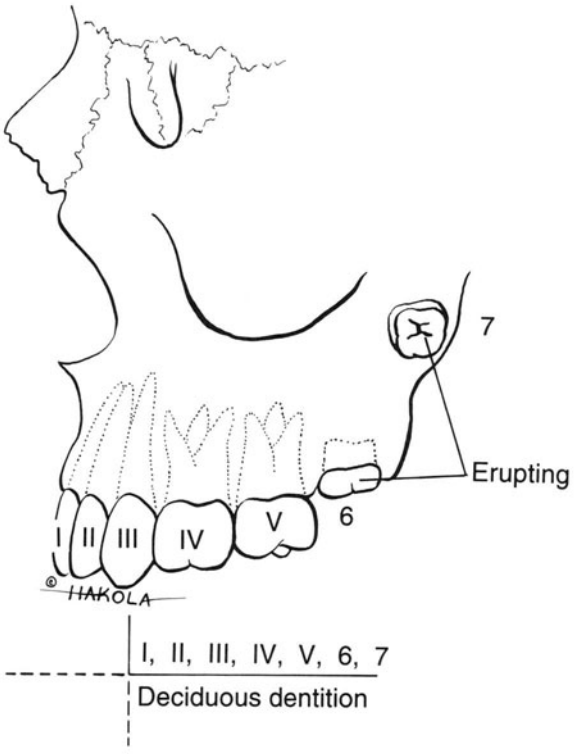


Figure 5.13. Upper left arch in a 5-year-old.

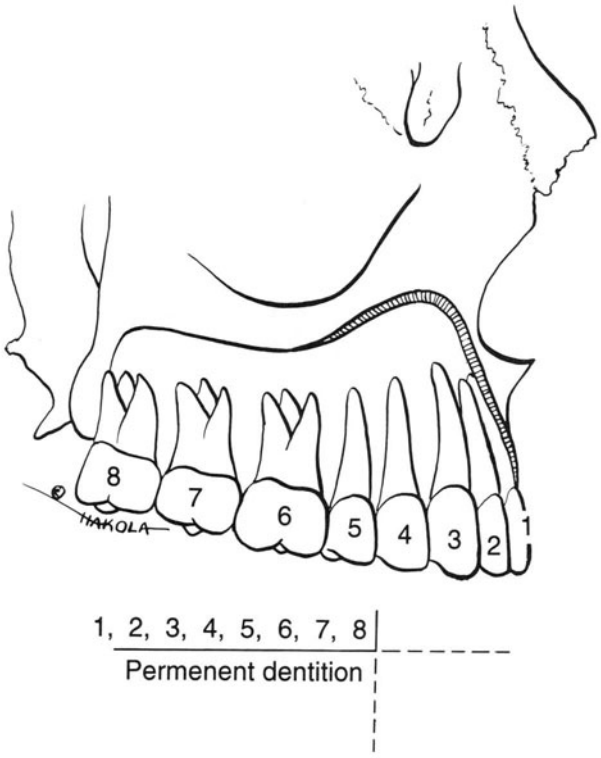


Figure 5.14. Upper right arch in a 22-year-old.

the patient's right upper quadrant. An example of how to represent these teeth individually in a shorthand form would be by representing the upper left quadrant and the number of the tooth as the upper left first bicuspid.[4] The upper right lateral incisor would be represented.[2]

Another method to denote the dentition in the arch is to use a formula where the teeth are numbered starting in the right upper quadrant and going forward all the way around the arch (1 to 18). The lower left third molar is now 19 and then around to the full compliment of teeth in the lower arch to the right third molar, which is 32. Here, for example, the lower left central would be 24 and the lower right cuspid would be 27.

In Table 5.3, the calcification and eruption sequence of the deciduous and permanent teeth is shown. It must be mentioned that this is only a guide because eruption patterns of human teeth vary considerably in time especially among the sexes. It is only those eruption times that are way outside the ranges that are considered as pathologic. The eruption of the deciduous teeth generally follows front to back with the upper centrals and laterals erupting first and the cuspids next, and on back to the molars erupting at age 3 years. The sequence in the permanent teeth is quite different. Here the upper and lower first molars are the first teeth to erupt into the arch. They position themselves just behind the deciduous second molars and are usually fully erupted by age 6 or 7. This is an important concept, for these teeth act as pillars, so that if the loss occurs before the dentin is completely erupted, a disturbance in the occlusion is likely to occur. This was very distinctly discussed by Dr. Angle in his landmark work in *Dental Cosmos*, 41:248, 1899. He states, "all teeth are essential yet in function and influence some are of greater importance than others, the most important of all being the first permanent molars." These teeth act to maintain the dynamic balance of the occlusion between the forces of the anterior arch with the forces of the molars on the posterior portion of the arch. Early loss of these teeth results in tilting of adjacent teeth, overeruption of the opposing teeth, and migration of the adjacent teeth into the first mandibular space creating marked dental disharmony.

An example of the importance in the pillar action of the first molar to maintain space can be seen in the "Leeway Space" of Nance, as described by Graber in his book *Orthodontics Principles and Practices*, J.B. Sanders Co. He states that the average combined width of the mandibular deciduous canine, first, and second molars is 1.7 mm greater than the permanent canine, first and second bicupsid. In the maxillary arch this overall width is only slightly wider (0.9 mm) than their permanent counterpart (Fig. 5.15). Thus, the loss of the pillar action of the first molar with subsequent drifting or tilting of the adjacent permanent teeth as they erupt could easily destroy this exact alignment.

Table 5.3. Calcification and eruption sequences of deciduous and permanent teeth.

Deciduous dental eruption schedule

	Calcification of crown begins	Tooth erupts	Calcification of root ends	Tooth sheds
Central Incisors	4 fetal months	6–7½ mos.	1½ years	7 years
Lateral Incisors	4½″ ″	7–9 ″	1¾ ″	8 ″
Canines	5 ″ ″	16–18 ″	3¼ ″	12 ″
First Molars	5 ″ ″	12–14 ″	2½ ″	10 ″
Second Molars	6 ″ ″	20–24 ″	3 ″	11 ″

Permanent dental eruption schedule

	Calcification of crown begins	Calcification of crown ends	Tooth erupts	Calcification of root ends
Central Incisors	3–4 months	4–5 years	6–8 years	9–10 years
Lateral Incisors	3–12 ″	4–5 ″	7–9 ″	10–11 ″
Canines	4–5 ″	6–7 ″	9–12 ″	12–15 ″
First Premolars	1½–2 years	5–6 ″	10–12 ″	12–13 ″
Second Premolars	2–2½ ″	6–7 ″	10–12 ″	12–14 ″
First Molars	At birth	2½–3 ″	6–7 ″	9–10 ″
Second Molars	2½–3 years	7–8 ″	11–13 ″	14–16 ″
Third Molars	7–10 ″	12–16 ″	17–21 ″	18–25 ″

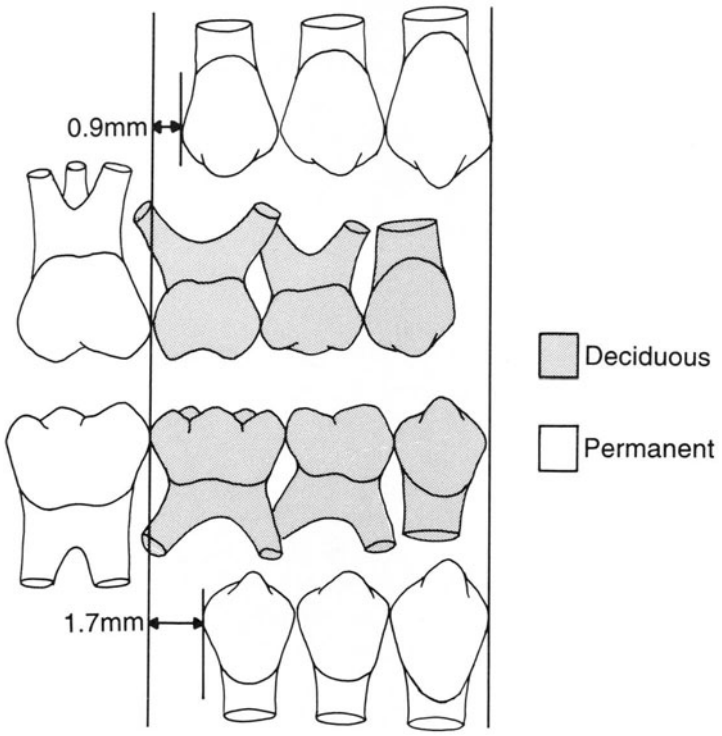


Figure 5.15. Leeway Space of Nance. In the maxillary arch, the combined width of the deciduous cuspid, first, and second molars is 0.9 mm wider than the width of the permanent cuspid, first, and second bicuspids. In the mandible, that Leeway Space is a slightly larger 1.7 mm, as described by Nance.

A Description of the Individual Teeth in the Right Side of the Dental Arch (Fig. 5.16)

Upper Central Incisor

This tooth has a broad crown and a shovel shape. When the tooth first erupts it, as well as the lateral incisors in both the maxillary and mandibular arches, have three rounded cusps called mamelons (see Fig. 5.17). These are quickly worn away with occlusal abrasion. It is important to note that if in an adult you still see mamelons present on the centrals and laterals you can assume that very little contact has been made between these teeth, as is frequently seen in Class III malocclusions or open bite deformities. The root of the fully formed central incisor is relatively short (about 12 mm) and cone shaped, and as a result can be pulled easily inferiorly with a wire or elastic traction. As a result interproximal wires should not be placed around this tooth and attached to an arch bar. This could easily result in partial extrusion of the tooth below the upper incisal plane. The same is true of the upper laterals and the lower central and lateral incisors.

Upper Lateral Incisor

This tooth is similar to the central but has a slightly narrower crown width mesially to distal. The root frequently has a slight distal bend at its apex. The root length is also about 12 mm.

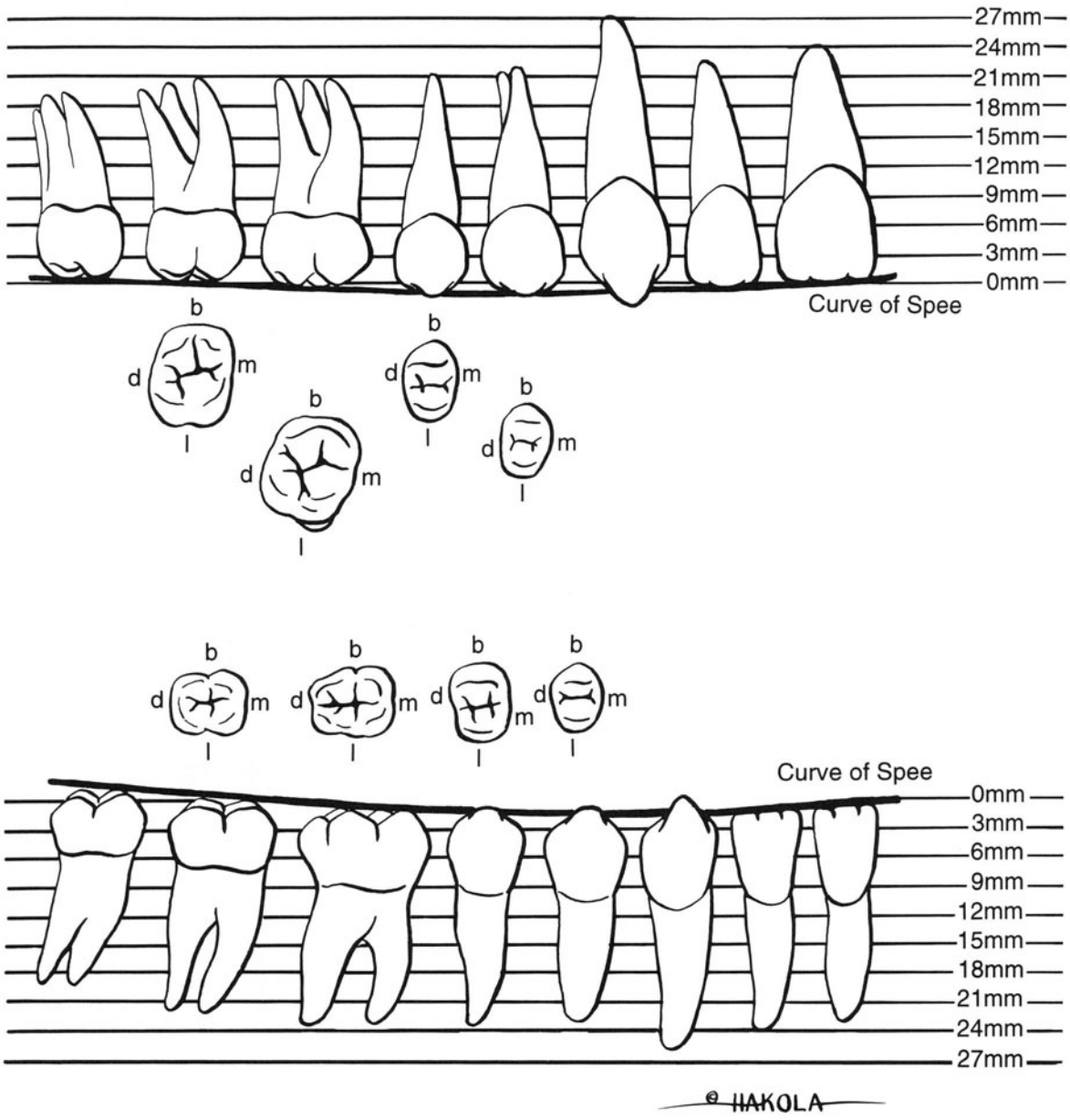


Figure 5.16. Anatomy of the teeth of the right arch.

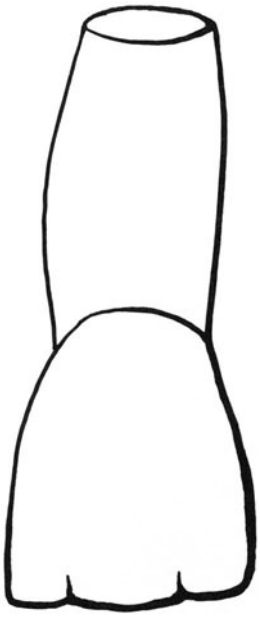


FIGURE 5.17. Upper left central of a child. Note the incompletely formed root and the three distinct cusplets or mamelons.

Upper Cuspid

Here, the incisal edge has a sharp point. It has a short blunted lingual cusp or cingulum. The root is longest and strongest in the dental arch (16 mm) with an overall tooth length of 27 mm. The apex of the root curves distally. This root structure is why this tooth should be included when placing arch bars. However, because of the low lingual crown anatomy (Fig. 5.18A), it is necessary to loop the arch wire around the arch bar to hold the wire as close to the neck of the tooth as possible. Figure 5.18B shows similar wires in the mandibular arch.

Upper First Bicuspid

These teeth are the first to have a true occlusal surface. The buccal cusp is similar to the cuspid but here the cingulum is now a fully developed lingual cusp. The root of the maxillary first bicuspid can be either one or two roots. Sicher states that over 50% of these teeth had two roots with two root canals.

Upper Second Bicuspid

This tooth is somewhat smaller than the first bicuspid. It has a lingual and buccal cusp of equal height. The root is rarely divided. It frequently has a deep groove and its root canals may be totally separate or may fuse at variable distances from the apex of the root.

Upper First Molar

Here the occlusal surface is rhomboid shaped. It frequently has an extra cusp on the mesial lingual surface called Carabelli's cusp. Sicher states it is present in 10–15% of upper first molars as a well-developed cusp, but a remnant such as a pit or groove may be present in the mesial lingual cusp in up to 40% of upper first molars. The roots are three, with the lingual (or palatal) and a distal and mesial root. It is important when extracting this tooth to "bring it out toward the buccal" in order not to fracture the palatal root, which converts a simple extraction to a very difficult one.

Upper Second Molar

The overall shape in this tooth is similar to the upper first molar except there is no Carabelli's cusp. The roots are also similar, except they do not diverge as much and

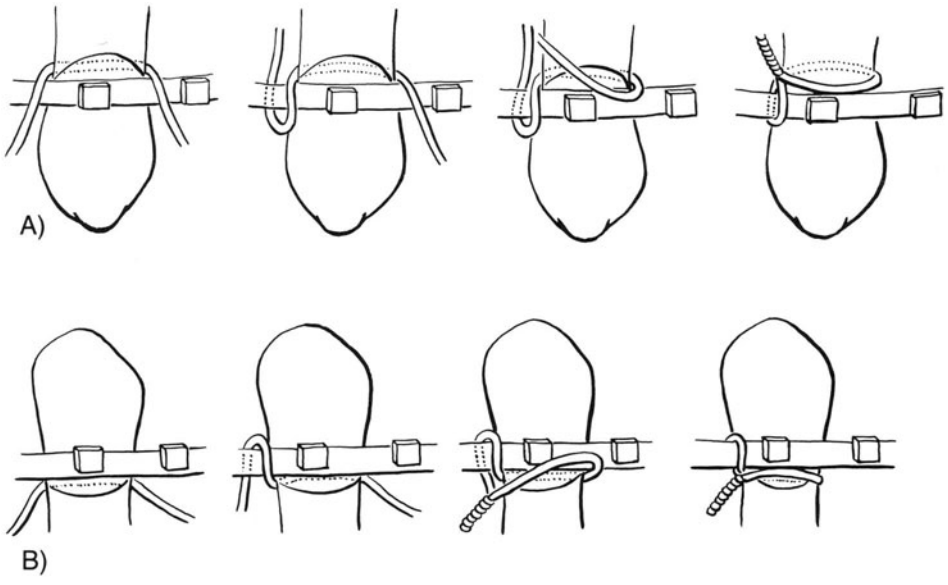


FIGURE 5.18. (A,B) This type of wire technique is used because the cuspids are conically shaped in order to have the arch bars hold on the necks of the teeth.

are more frequently fused into one mass but still maintain the three separate root canals.

Lower Central Incisor

The crown of this tooth is narrow with a sharp mucosal edge. As with the upper central incisors, there are mamelons present in the newly erupted lower central incisors that quickly are worn away with incisal function. The root is straight and the overall tooth size is quite small, and therefore it too should not be attached to an arch bar because it could easily be avulsed from its socket.

Lower Lateral Incisor

This tooth is very similar to the central incisor, only it's a little longer.

Lower Cuspid

This tooth is smaller and shorter than the upper cuspid (overall length 25 mm vs. 27–28 mm for the upper cuspid). Because of its similar shape to the upper cuspid it must also be wired to the arch bar with a loop wire (Fig. 5.18A and 5.18B). The root is shorter than the upper cuspid and not as strong, but it should be used to help retain the lower arch bar because it does have good retentive strength.

Lower First Bicuspid

Here there are two cusps, a buccal and lingual, but when compared with the upper first bicuspid the lingual cusp is smaller and shorter. The root here is singular with one root canal.

Lower Second Bicuspid

The crown of the lower second bicuspid is larger than the first with a larger, more developed, lingual cusp. Its root is also singular with a root canal, but is longer and stronger than the lower first bicuspid.

Lower First Molar

This tooth has five cusps, three on the buccal and two on the lingual. It is rectangular shaped. The lingual cusps are slightly higher than the buccal ones because they fit into the central fossa of the upper first molar. There are two roots, a distal and mesial root with two canals, and these roots frequently deviate distally in the arch.

Lower Second Molar

The crown of this tooth is typically described as a "hot cross bun" with two lingual and two buccal cusps. Both are of equal height. The roots also are two, a mesial and distal root with two root canals.

Eruption of a Tooth

The eruption of a tooth cannot be compared to the growth of a plant out of the ground. This is because the growth of the plant pushes against an inert element, the earth alone, whereas the growth of a tooth occurs in living bone. When there is pressure exerted in living bone it responds either by osteoblastic proliferation and more bone formation or osteoclastic activity and bone resorption. As the tooth develops in its dental sac, there is a resorptive surface at the periphery of the dental sac that permits the growth and development of the crown of the tooth. This is called the preeruptive phase of eruption. With the development of the root the active phase of eruption begins. As the root grows down into the alveolar bone the resultant force is upward in the mandible and downward in the maxilla. This process of eruption proceeds as the tooth emerges through the mucosa and continues until the erupting tooth comes in contact with the tooth of the opposite arch. Eruption also occurs in a rotational axis or in a transverse labiolingual or buccolingual axis (tilting or tipping of teeth). Finally, a tooth may move parallel to its long axis or drift, as can occur when an adjacent tooth is absent and the tooth migrates into the adjacent tooth space. This frequently occurs in the molar region and is associated with tilting (such as a second molar migrating and tilting into a space created by the absent first molar).

Once the tooth is fully developed it still can erupt if, for example, it has no opposing tooth. It is believed that the negative force created by the unopposed tooth results in bone apposition at the apex of the tooth and facilitates the eruption until opposite contact is made.

The preeruptive movements of the permanent teeth are in principle the same as those of the deciduous teeth. There, developing dental sacs start out lingual to the erupting deciduous teeth. By the process of eccentric growth of the dental follicle, as stated by Sicher, these tooth germs position themselves in a staggered fashion in the arch as seen in Fig. 5.19A and 5.19B.

The larger size of these developing teeth results in a crowded position in the growing but relatively small jaw of the 5-year-old child, as is seen in Fig. 5.20. Thus, the follicles of these permanent teeth may be rotated or staggered in the arch and frequently are just above the inferior border of the mandible or just below the orbital floor in the maxilla. The permanent bicuspid crowns are smaller than the overlying deciduous molars and develop immediately below the bifurcation of these molar roots.

The permanent molars' position and growth is dependent on the forward growth of the mandible. This was discussed in the previous chapter on skull growth and is also seen in Fig. 5.20A and 5.20B. When the permanent molars begin their development they are in the base of the ascending ramus of the mandible, as seen in Fig.

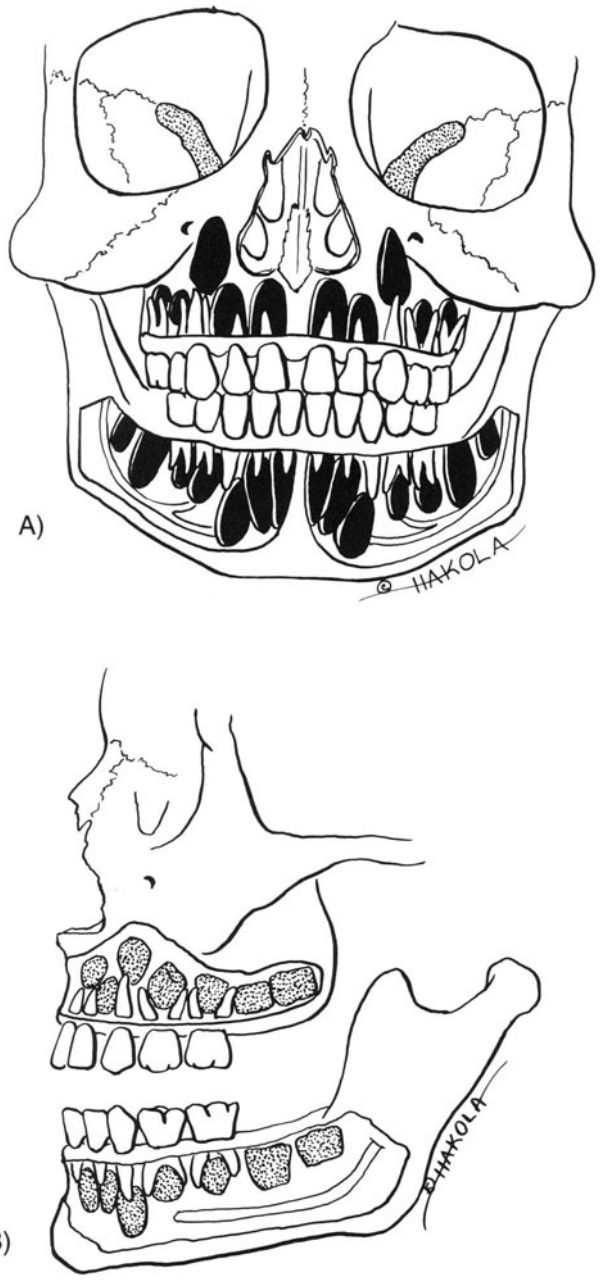


Figure 5.19. Skull of a 5-year-old showing position of the erupting teeth in the maxilla and mandible. Note the close approximation to the infraorbital foramen and the orbital floor in the maxilla and the inferior border of the mandibular body.

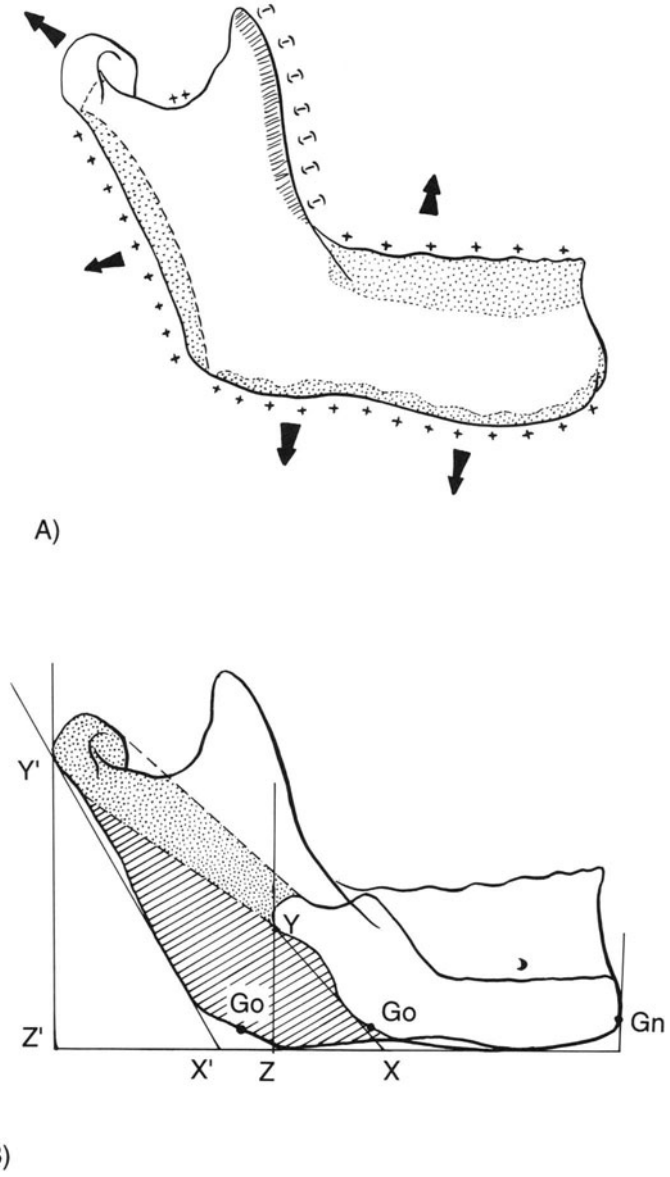


Figure 5.20. (A) Appositional growth of the posterior border of the mandible, alveolar ridge, inferior body of the mandible with interstitial and appositional proliferation at the condyle. This results in increased dental arch length with room for the erupting molar teeth. (B) Diagram showing infant and adult mandibular growth superimposed at the gnathion. Stippled area indicates cartilaginous growth at the condyle and hatched area represents appositional growth at the ramus.

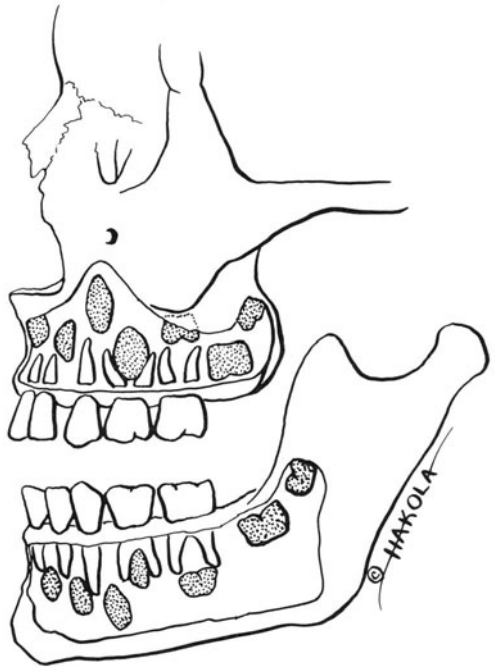


Figure 5.21. Side view of a child at 4 years of age.

5.21. The developing upper molars occur in the expanded maxillary tuberosity (Fig. 5.21). Thus, one can see that if horizontal growth of the mandible doesn't occur or the maxilla is hypoplastic these molar teeth may be unable to erupt because of "block out" by the erupted adjacent teeth. They therefore remain in the bone obliquely or even in a horizontal position.

The position of the developing and erupting teeth as seen in Fig. 5.19 also demonstrate how difficult reduction of fractures of the maxilla and mandible are in the young child. There is little or no room to place plates and screws without risking injury to these developing teeth. An osteotomy of the maxilla would also be extremely difficult because of the absence of the maxillary sinus and the high position of the developing cuspid and bicuspids in relation to the orbital floor.

Maxillary Sinus

The maxillary sinus at the time of birth is very small (about the size of a pea) (see Fig. 5.22). As the face grows and the teeth erupt the sinus enlarges in an inferior direction. This may explain why the maxillary sinus ostium is at the upper end of the adult maxillary sinus cavity. In most individuals, Sicher states the maxillary sinus continues to expand throughout life. This expansion penetrates deeper into the alveolar process and thus we frequently see the apices of the molar teeth sticking up into the sinus floor. It has been said than on occasion the maxillary sinus may even invade into the body of the zygomatic bone. The enlargement of the maxillary sinus explains in part why maxillary fractures are less often seen in children than in adult patients. Also, the intimate relationship of the sinus to the roots of the bicuspid and molar teeth explains why with maxillary sinusitis one can have a feeling of pressure or pain referred to these teeth.

Occlusion

Curve of Spee

When looking at the maxillomandibular occlusion from the buccal surface an anterior position curve is noted. This curve is called the Curve of Spee, named after Van Spee from his book on *Prosthetic Dentistry* in 1928. It extends from the tip of the mandibular cuspid and follows along the buccal cusps of the mandibular posterior teeth (see Fig. 5.23A).

This curve is usually slight and only in a few patients does it have a pronounced arc. It's believed to be an adaptation of the teeth to a balanced occlusion and it is caused by the tendency of a single tooth to assume a position in the jaw where its long axis is best suited to the forces of mastication. The relation of the long axis of the upper and lower arches form reciprocal curves (see Fig. 5.23B), which are convex in the maxilla concave in the mandible. This position gives each tooth the optimal resistance under maximal force from the muscles of mastication. The obliquity of the resultant force produces an angular position of the teeth in the arch, is affected by the arc of rotation and angulation of the condyle in the glenoid fossa, and the forces of the muscles of mastication. This curve permits the maximum utilization of tooth contacts during function that would not be possible if there was a flat occlusal plane.

The term articulation as defined by *Dorland's Medical Dictionary* is the union or junction of two bones in the skeleton, and here could easily refer to the condyles relationship in the glenoid fossa. It also refers to the contact relationships of the occlusal surfaces of the upper and lower teeth during movements of the lower jaw in mastication. This is opposed to occlusion, which is the relation of the upper and lower teeth to each other after closure of the jaws.

The teeth are not in continual contact in normal individuals. Instead there is a position the mandible assumes when the patient is erect, and the muscles of mastication are relaxed. This is called the mandibular rest position. Here the teeth are not in contact but have a space of 2–3 mm between them, the freeway space. When the patient is in rest position, even though the muscles are relaxed, there is still some muscle activity

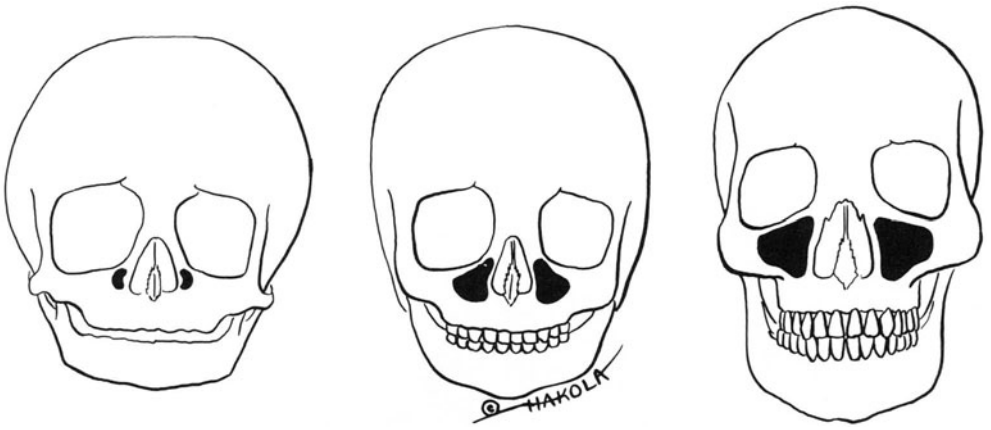


Figure 5.22. Development of maxillary sinus from infant to adult.

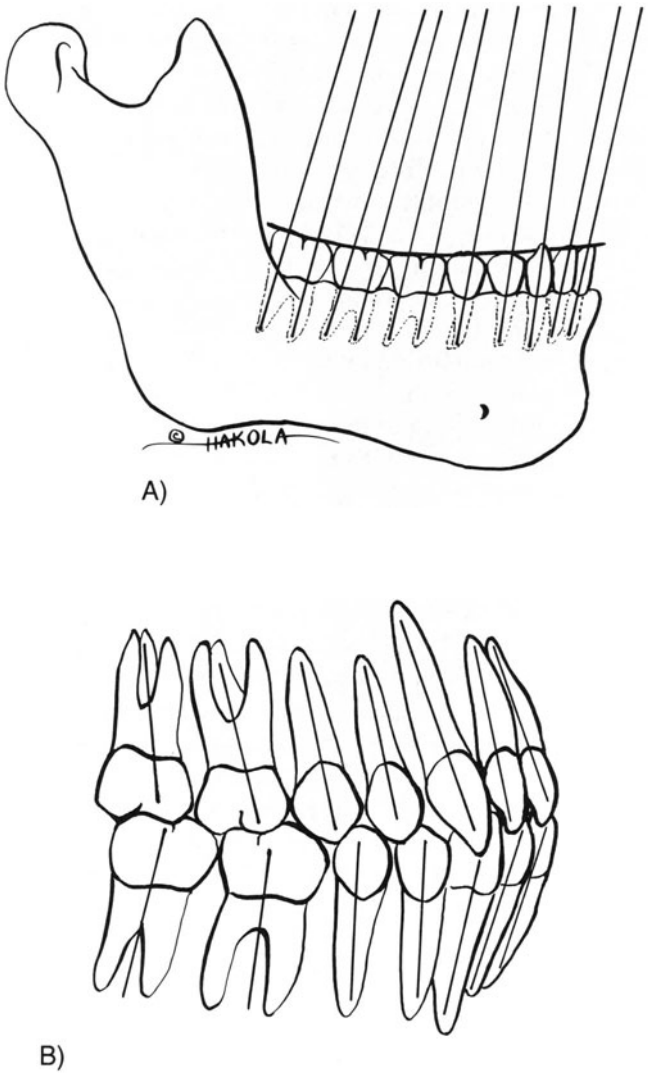


Figure 5.23. (A) Angulation of the lower teeth. (B) Angulation of the upper and lower teeth.

present, the muscle tonus, which is present throughout the body unless the patient is paralyzed under general anesthesia. This freeway space is important and must be maintained. If the bite is artificially "opened" and exceeds the freeway space distance, the muscles of mastication, especially the external pterygoid, become chronically stretched with an increase in tonus. This can result in fatigue and increased muscle irritation, with muscle spasm and pain.

Centric Relation and Centric Occlusion

Generally speaking, centric relation is the most retruded unstrained position of the mandibular condyle in the glenoid fossa. This is also called terminal hinge position. Centric relation does not in itself indicate occlusion of the teeth, it is simply a condylar glenoid fossa relationship. Centric occlusion, on the other hand, is the maximal contact of the incline planes of the opposing cusps of the mandibular and maxillary arches. In this relationship of maximal contact there must be bilateral symmetrical contact and a balanced and unstrained relationship of the condyle in the glenoid fossa. In other words, in the ideal situation where there is centric occlusion there must also be centric relation. If, however, there are present premature cuspal contacts as the jaws are closed (loss of teeth with opposing overeruption of teeth from the upper arch toward the mandible, restorations with too high an occlusal contact, and malpositioned teeth), the centric occlusion can be destroyed. Instead there is with jaw closure a premature contact of the teeth and a slide into occlusion. This can distract the condyle either anteriorly or too far posteriorly within the glenoid fossa, thus destroying the unstrained position (centric relation). With the loss of centric relation there can be mus-

cle imbalance with increased muscle tonus, muscle overactivity, spasm, and pain. This is the basis for occlusal equilibration to reestablish a harmonious occlusion and the basis of orthodontics and orthognathic surgery to reestablish normal jaw relationship so that when there is centric relation of the condyle in the glenoid fossa there is also centric occlusion of the dentition.

One other term must also be discussed, and that is habitual occlusal relationship. This is the habitual occlusion a patient goes into when they occlude their teeth several hundred times a day while swallowing, talking, and so forth. It is imperative for dental and temporomandibular joint health that the habitual occlusion and centric occlusion position be the same so that there is harmony between the occluding teeth and the condyle in the glenoid fossa.

Overbite, Overjet, and Crossbite

The normal interarch relationships of the anterior teeth show two different types of overlap—a vertical and a horizontal overlap.

The horizontal distance from the labial incisal edge of the lower central incisor to the labial incisal edge of the upper central incisor when the jaws are in centric occlusion is called overjet. In a Class II malocclusion, as in Fig. 5.24, there is frequently an increased amount of overjet. (This may not be the case in Class II, Division 2, and cases with deep bites). In the vertical plane the vertical overlap is called overbite and is represented by the distance from the incisal edge of the upper central incisor to the incisal edge of the lower central incisor while the teeth are in centric occlusion. Looking at Fig. 5.24, a Class II, Division 2 case with a deep bite will have an increased overbite whereas an end to end bite (Class III) will have no overbite whatsoever. Some patients may even have a distinct gap between the incisal edges of the central incisors when the molars are in centric occlusion. This is then called an anterior open bite deformity.

In some occlusions, especially significant Class III malocclusions, the normal relationship of the upper dentition being labial to the lower dentition is reversed. This situation is called an anterior crossbite.

In the occlusion of the posterior teeth there can also be an overjet, overbite, open bite, and crossbite. Figure 5.25A shows the normal buccolingual arch relationship of the mandibular and maxillary molar teeth. Here we see that the mandibular buccal cusps occlude in the central fossae of the maxillary molars and the maxillary lingual cusps articulate in the central fossae of the mandibular teeth. This is explained because in the normal individual the total length of the maxillary arch is 128 mm, whereas the mandibular arch is only 126 mm. (The maxillary molar buccal cusps also overlap the mandibular molar buccal cusps as seen in Fig. 5.25B.) This produces some degree of overjet and overbite in this molar region. If there were a gap between the maxillary and mandibular occlusion then a lateral posterior open bite could exist as well.

When the more buccal position of the maxillary teeth versus the mandibular teeth is reversed in the molar occlusion, then a posterior crossbite can exist (Fig. 5.25C). It may be bilateral or unilateral. In this type of malocclusion, the mandibular lingual cusp articulates in the central fossa of the maxillary teeth and the maxillary buccal cusp articulates on the central fossa of the mandibular teeth.

The overlap of the maxillary dentition over the mandibular teeth produces a definite advantage in the chewing of food. The overlap of the maxillary molars prevents the cheek from being entrapped and "chewed on" when the molar teeth occlude. Also as the teeth are brought into contact the majority of the food is shunted toward the tongue, which efficiently returns the food back to the occlusal table to be repulverized in the chewing process.

The differences in the mesiodistal diameters of the upper and lower incisors cause a distal position of the upper cuspids, bicuspids, and molars to their counterpart in the lower arch. Thus, the upper cuspid occludes distal to the lower cuspid, between it and the lower first bicuspid. In the molar region this distal position of the upper dentition results in the mesial buccal cusp of the upper first molar to articulate with the buccal groove of the lower first molar (see Fig. 5.26), which is the basis for Angle's classification of occlusion.

Classification of Malocclusion

Malocclusion can be divided into dental dysplasia, skeletodental dysplasias, and skeletal soft tissue dysplasias. As we learned earlier in this text, Angle felt that the upper first molar was the keystone of the arch and as a result the "key to occlusion." Angle's classification serves as a tool to describe the anterior–posterior relationships of the maxillary and mandibular dental arches. Using it makes it possible to scientifically categorize malocclusions and gives us "common ground" to communicate information to our colleagues either locally or in other countries. It has more recently been expanded to include skeletal and soft tissue relationships of the upper and lower jaws.

Dental dysplasias exist when individual teeth within one or both jaws are abnormally related to each other. Thus we see as described earlier in this section that dental dysplasias may be due to lack of space to accommodate the teeth. They may also be due to congenitally missing teeth, which in decreasing frequency are the maxillary lateral incisors, the mandibular second bicus-

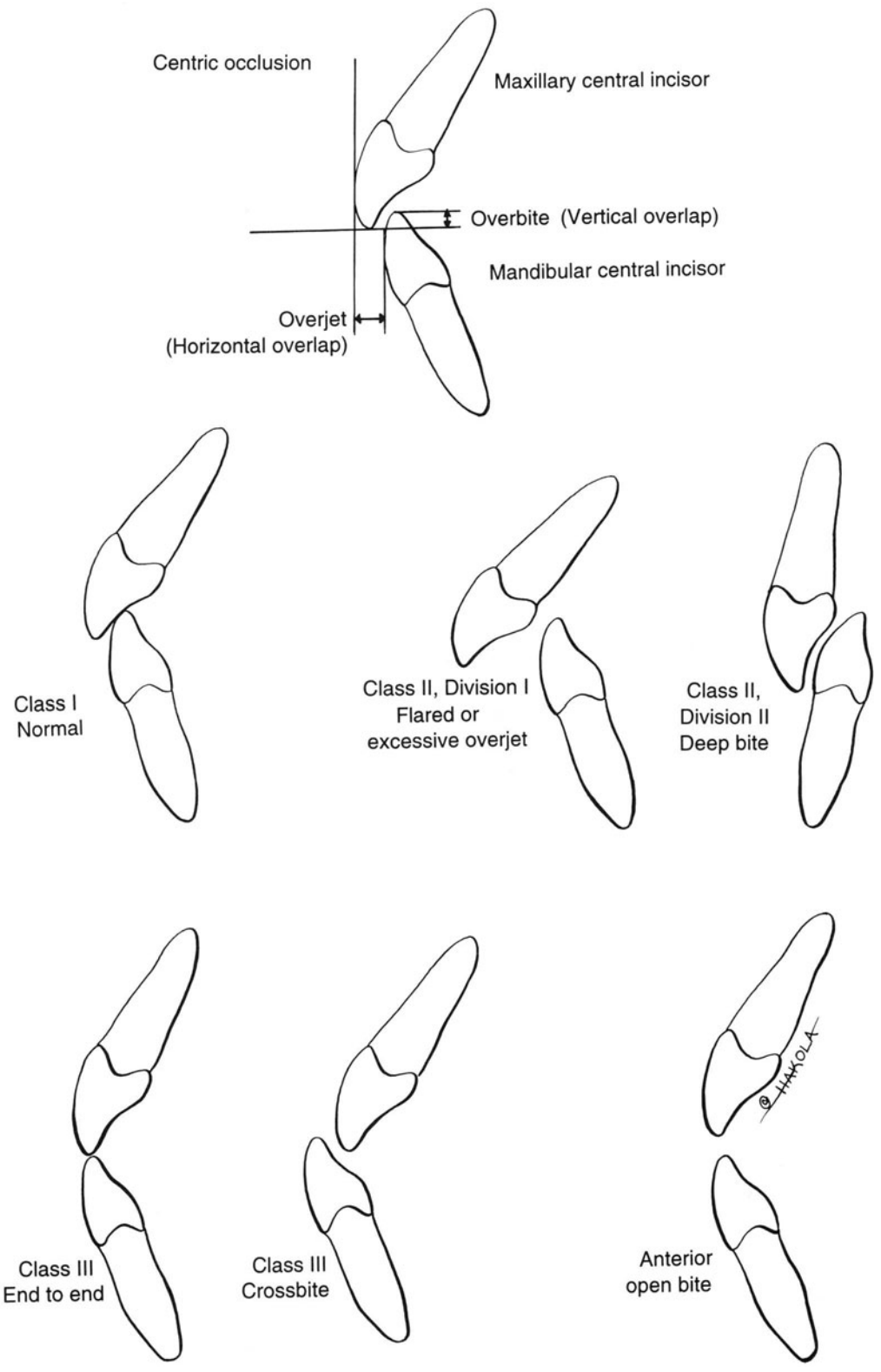


Figure 5.24. The relationship of the maxillary and mandibular central incisors to each other in normal situations and in instances where there is an anterior jaw discrepancy.

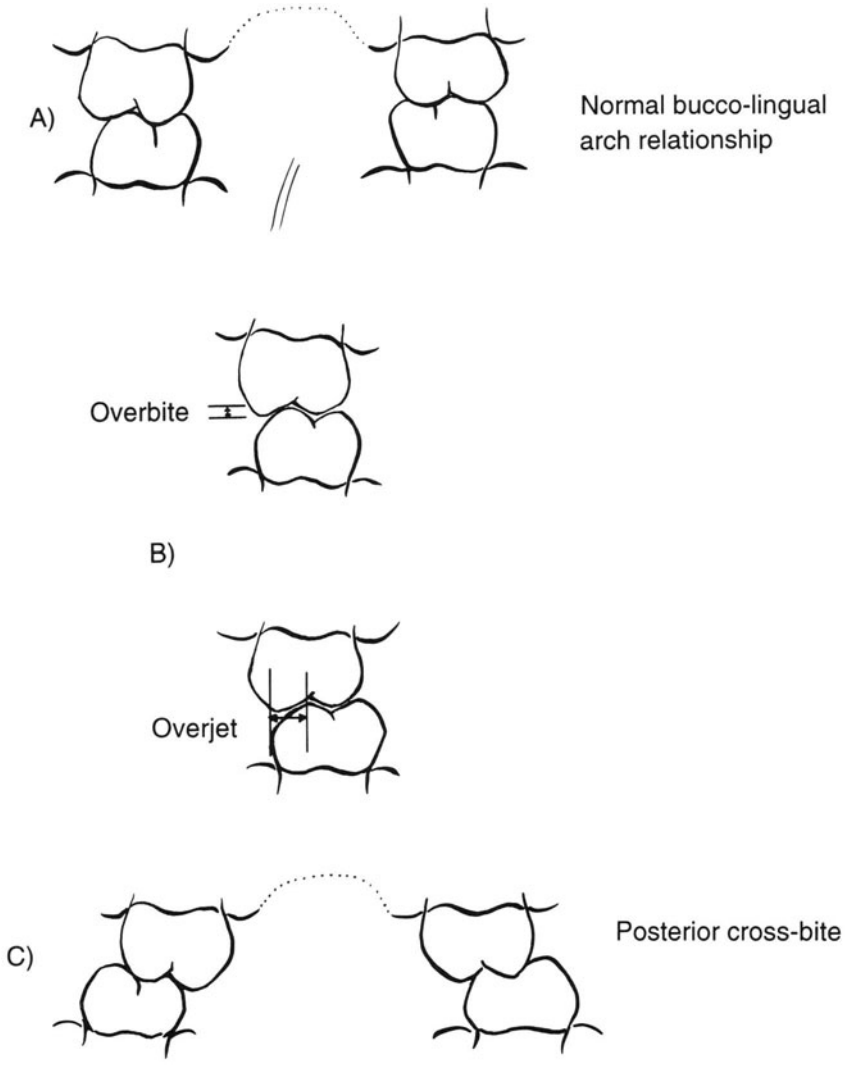


Figure 5.25. Variations in the molar region.

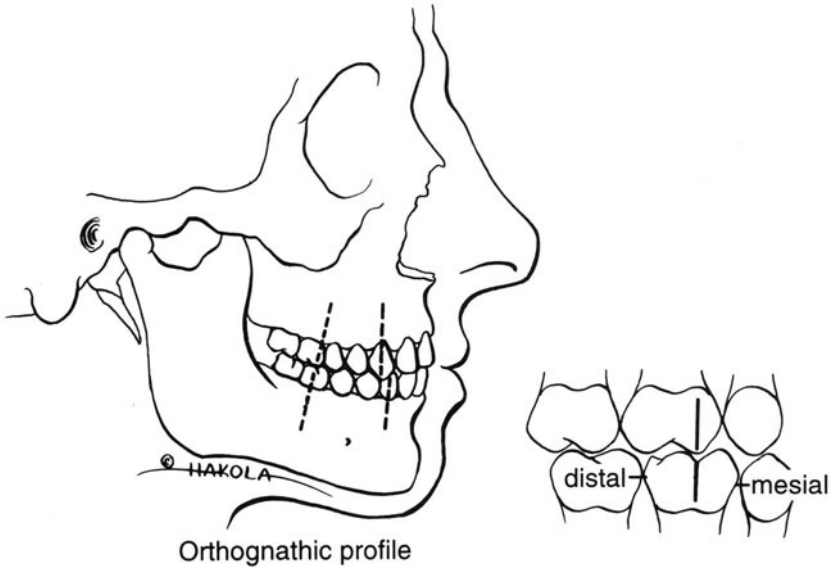


Figure 5.26. Neutroocclusion Class I profile.

pids and the maxillary and mandibular third molars. It is also possible to have extra- or supernumerary teeth producing dental dysplasias, and these may even be present in the same arch as in a patient with congenitally missing teeth. Other causes of dental dysplasias may be due to premature loss of deciduous or permanent teeth or prolonged retention of deciduous teeth, thereby disrupting the orderly eruption of the permanent teeth.

However, the most likely cause of malocclusion is the patient's basic hereditary or ethnic pattern, which results in discrepancy in the size and number of the teeth in relation to the size and shape of the skeletal support of the jaws. In a large study of 21,328 children, 6–18 years of age, by Dr. Woeffel in the United States, 71.7% had dental malocclusions whereas 28.3% had an acceptable occlusion. When classified according to Angle's classification: 28.3% had Class I occlusions, 22% had Class II occlusions, and 5.7% had Class III occlusions. In contrast, abnormal jaw relationships and malocclusions in Swedish children were found by Seipel, in a study at the State Institute of Human Genetics and Race Biology in Uppsala, Sweden, to be between 18 and 20%. Other studies comparing ethnic differences contrasted the 22% of United States children who had Class II malocclusions to that of Japanese children who had a low incidence of Class II malocclusions, but a high incidence of mandibular prognathism or Class III malocclusions, and Eskimo children, in whom any malocclusion is seldom seen. (Graber—*Orthodontics Principles and Practice.*)

Hereditary influence is also seen when comparing Japanese and American children. The evidence of Class II and Class III malocclusion in American children is 22% and 5%, respectively, almost reversed when comparing the Class II and Class III occlusions in Japanese children who have a much higher incidence of Class III malocclusions. In cultures such as the Eskimo, any type of malocclusion is seldom seen.

If in addition to irregularly positioned teeth there is a distorted relationship between the maxilla and the mandible to the cranial base, one has a skeleto-dental dysplasia. This is seen in Aperts and Crouzon Syndrome or in cases of fibrous dysplasia, where excessive skeletal overgrowth can occur producing secondary dental and skeletal abnormalities. In certain cases the soft tissues may be overgrown as with the patient with an excessive soft tissue chin button or absence of soft tissue and hard tissue structures as in hemifacial microsomia. Here the occlusion may be easily corrected, but consideration must also be given to change in the soft tissues to produce an aesthetically harmonious final result.

In discussing Angle's classification it is important to realize that Angle was describing different forms of malocclusion. He divided malocclusions into three broad classes dependent on the relationship of the upper and lower first molars: Class I neutroocclusion, Class II distoocclusion and Class III mesioocclusion.

Class I (Neutro-Occlusion)

Note that, as in Fig. 5.26, the anterior–posterior relationship of the maxillary and mandibular molars is in neutral position (mesiobuccal cusp of the maxillary first molar articulating with the buccal groove of the lower first molar), but the anterior occlusion has some forms of dental abnormality with the anterior teeth flaring, being crowded, or having excessive lingual tilting. It is also possible to see a Class I molar and cuspid relationship but have the entire upper and lower dentition positioned forward on their alveolar bases (called bimaxillary protrusion). Another possibility is having the posterior molar relationship Class I but the anterior teeth and bicuspids out of contact, producing an anterior open bite. (It is important to remember that an open bite may also occur in Class II and III malocclusions as well.) The effects of these malpositioned teeth on the soft tissues may produce excessive eversion of the vermilion, producing lips in the bimaxillary protrusive case or the lip incompetence that is so frequently seen in anterior open bite cases.

However, although Class I classification as described by Angle takes into account anterior discrepancies, in current discussions, the term Class I relationship has become known as the "ideal" when discussing skeletal, soft tissue and dental relationship of the face and jaws.

Class II (Disto-Occlusion)

Here, the lower arch is in a distal or posterior position in relation to the maxillary arch. The mesial buccal cusp of the maxillary first molar now articulates with the distal portion of the mandibular second bicuspid and the mesial cusp of the first molar. The remaining teeth reflect this distal position of the mandible. In fact, Angle subdivided this Class II into Division I and Division II depending on what the relationship of the anterior teeth were to each other.

Division 1 As seen in Fig. 5.27A, the molar relationship is as described above but the anterior teeth have a tendency to flare, producing a significant overjet. This results in muscle imbalance with loss of upper and lower lip competence. In this division the lower lip frequently becomes everted because of its contact with the flared maxillary central and lateral incisors. Frequently the arch form is not u-shaped but instead is v-shaped because it is narrowed in the cuspid and bicuspid region. This narrowed maxillary arch tends to constrict the tongue, which then puts pressure on the anterior teeth with swallowing and thereby accentuates the labial flaring and excessive protrusion of the anterior maxillary teeth. As stated by Graber (*Orthodontia Principles and Practice*), research on growth and development and numerous cephalometric studies point to the strong influence of hereditary patterns as modified by compen-

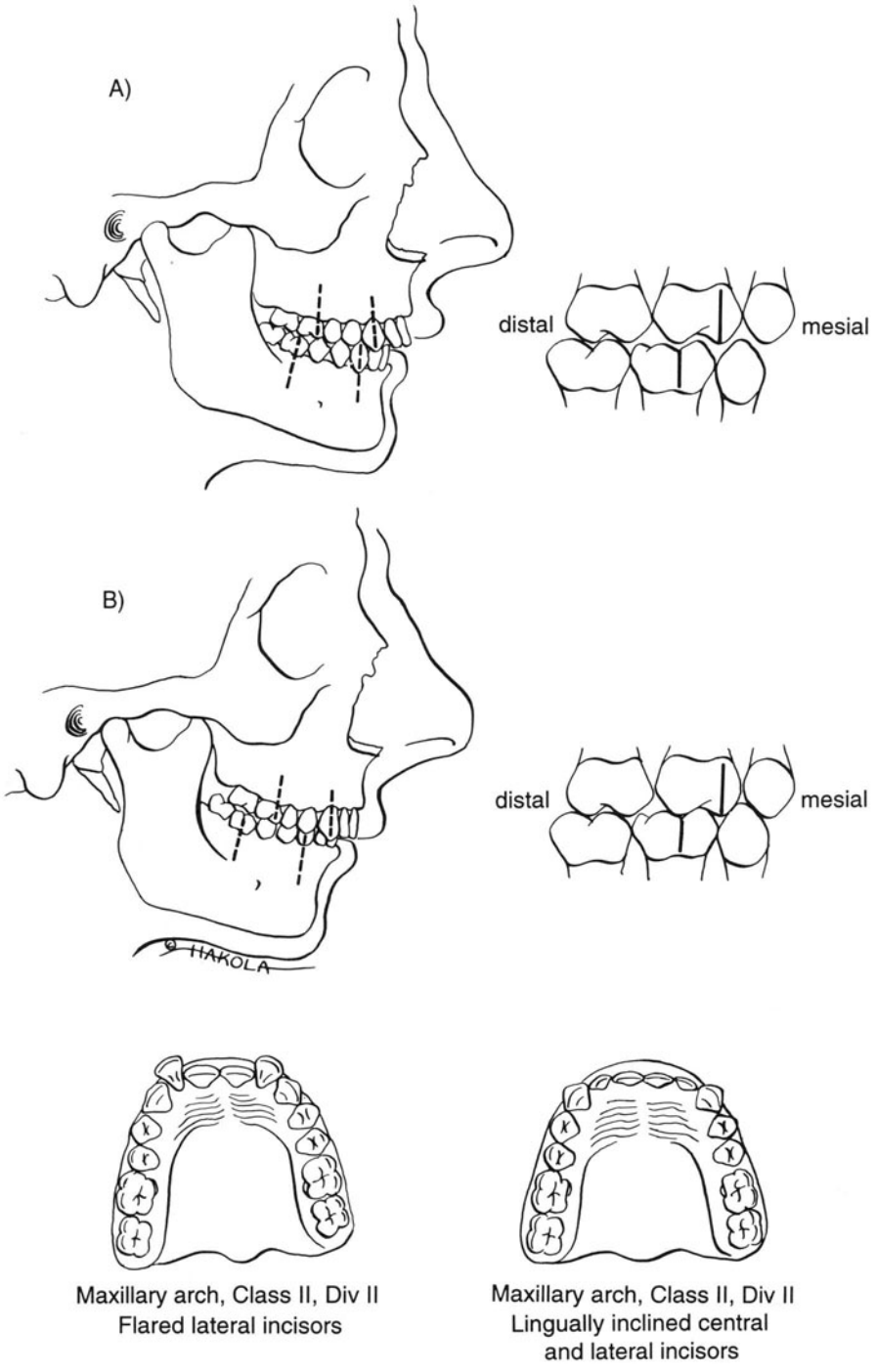


Figure 5.27. Retrognathic or Class II profile. (A) Div I. (B) Div II.

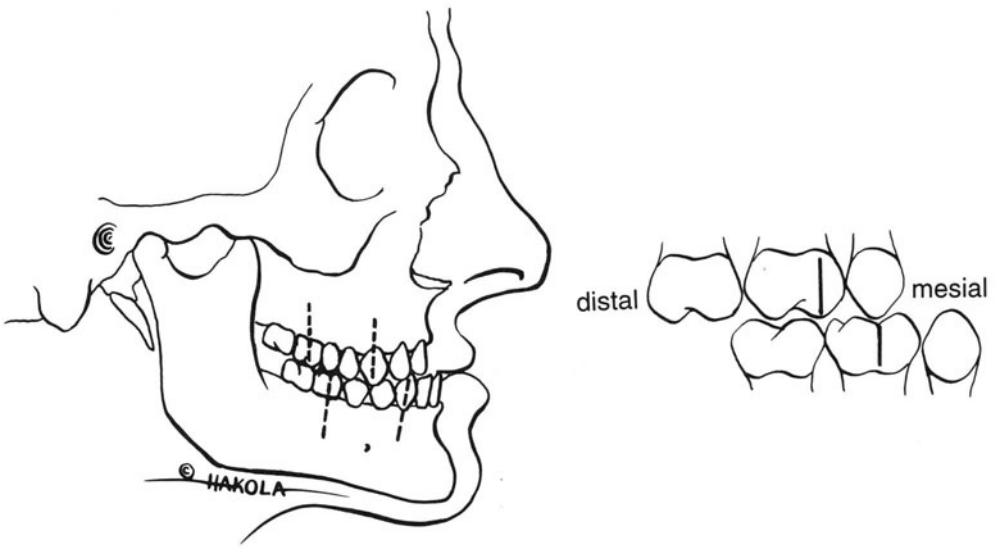


FIGURE 5.28. Prognathic or Class III profile.

sating functional factors as a basis for most Class II, Division I malocclusions.

Division II As seen in Fig. 5.27B, in this division again the mandibular molar relationship is Class II or in disto position when compared to the maxillary dentition. The anterior dental relationship, however, is quite different here when compared to the Division I patient. The maxillary dental arch is not narrow but instead may be quite wide. There is an excessive curve of Spee with super eruption and lingual tilting of the lower anterior incisors. This produces a deep bite that may result in trauma to the maxillary palatal gingiva in excessive cases. The maxillary lateral incisors are either flared or in lingual version also. Because this is a tight deep bite the forced lingual tilt of the lower anterior incisors may cause their apices to protrude through the labial plate of bone. This tight locked bite can also force the mandible and its condyle into a retruded position in the glenoid fossa, which may lead to symptoms of temporomandibular joint pain, frequently associated with deep bite patients.

Class III (Mesio Occlusion)

In this type of malocclusion (see Fig. 5.28) the mandibular dentition is mesially positioned when compared to the maxillary dentition. Thus the mandibular first molar articulates with the maxillary first and second bicuspid teeth. In the anterior there is either an end-to-end bite or a crossbite relationship. The lower incisors in true prognathism are usually lingually displaced because of the increased mandibular length and forward mandibular position. This results in an increased lingual pull by the muscles of the lips on these anterior mandibular incisors, resulting in their lingual tilt. At the same time there is a decreased force by the tongue labially because it lies easily in the floor of the mouth, which is larger because of the increased mandibular length. The lingual tilt of the lower incisors and the labial tilt of the maxillary incisors seen in this class of malocclusion are called dental compensation. These dental compensations must be overcome orthodontically before surgery is performed to correct this malocclusion. In some instances the occlusion may give the appearance of a Class III relationship but is due entirely to a small hypoplastic maxilla. Here the mandibular length is normal but the excessively small maxilla produces a pseudo-Class III or pseudoprognathism. The patient has a flat canine fossa and a concave facial profile. The treatment in these cases is directed toward the maxilla with a maxillary advancement. If these cases were mistakenly treated in the mandible the result would be disastrous, producing a very flat facial profile and a very unhappy patient.

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CHAPTER 6

Local Anesthesia and Infiltration Techniques

James W. Ferraro and Irving M. Polayes

The fifth cranial nerve or trigeminal nerve is a mixed nerve, made up of a large sensory portion and a smaller motor portion. The sensory portion has a large half moon-shaped ganglion—known as the semilunar, Gasserian, or trigeminal ganglion—which fills the trigeminal groove in the floor of the middle cranial fossa. Three large divisions arise from this ganglion: first division—the ophthalmic nerve (V_1); second division—the maxillary nerve (V_2); and third division—the mandibular nerve (V_3).

The Ophthalmic Nerve V_1—Sensory Only

The ophthalmic nerve enters the orbit through the superior orbital fissure, where it divides into three branches. (1) The lacrimal nerve supplies branches to the lateral conjunctiva, a small area adjacent to the skin of the lateral canthus of the eye and to the lacrimal gland. (2) The nasociliary nerve, which enters the orbit after passing through the bifurcation of the lateral rectus muscle at the conus. It gives off branches: the posterior ethmoidal nerve, which is sensory to the sphenoid and ethmoid air cells, the anterior ethmoidal nerve, which is sensory to the dorsum of the nose after passing through a slit adjacent to the crista galli in the anterior cranial fossa, the short ciliary nerve, which carries postsynaptic sympathetic fibers to the dilator papillae muscle. It then terminates as the infratrochlear nerve, which is sensory to the conjunctiva of the medial caruncle and conjunctiva and skin of the medial aspect of the lower lid. (3) The frontal nerve runs forward just below the roof of the orbit and divides into the supraorbital and supratrochlear nerves, which innervate the skin of the upper eyelid, the forehead, and the scalp back to the occipital region (Fig. 6.1).

The Maxillary Nerve V_2—Sensory Only

The maxillary nerve passes through the foramen rotundum and enters the pterygopalatine fossa where it gives off the first of its seven main branches: (1) the meningeal branch to the meninges; and (2) the communicating branches to the sphenopalatine ganglion (Fig. 6.2A and 6.2B). From the ganglion, the greater and lesser palatine nerves emerge, which reach the hard and soft palate through the greater and lesser palatine foramen. These nerves supply the mucous membranes of the hard and soft palate and the palatal aspect of the gingiva. Also coming from the sphenopalatine ganglion are the posterior nasal branches, which supply the mucous membrane lining of the posterior inferior part of the nasal cavity. One of these posterior nasal branches, the nasopalatine nerve runs forward and downward along the nasal septum to the incisive foramen, where it supplies branches to the anterior part of the hard palate and the adjacent area of the gingiva (Fig. 6.2B and 6.2C). (3) Next to come off is the zygomatic nerve, which enters the orbit through the inferior orbital fissure to pass along the lateral wall of the orbit (Fig. 6.1) and divides into the terminal zygomatico-facial and zygomatico-temporal branches that supply the skin of the anterior temporal area and the lateral corner of the eye. (4) Just before the maxillary nerve enters the maxillary groove by passing through the inferior orbital fissure, it gives off the posterior superior alveolar nerve. This nerve runs down

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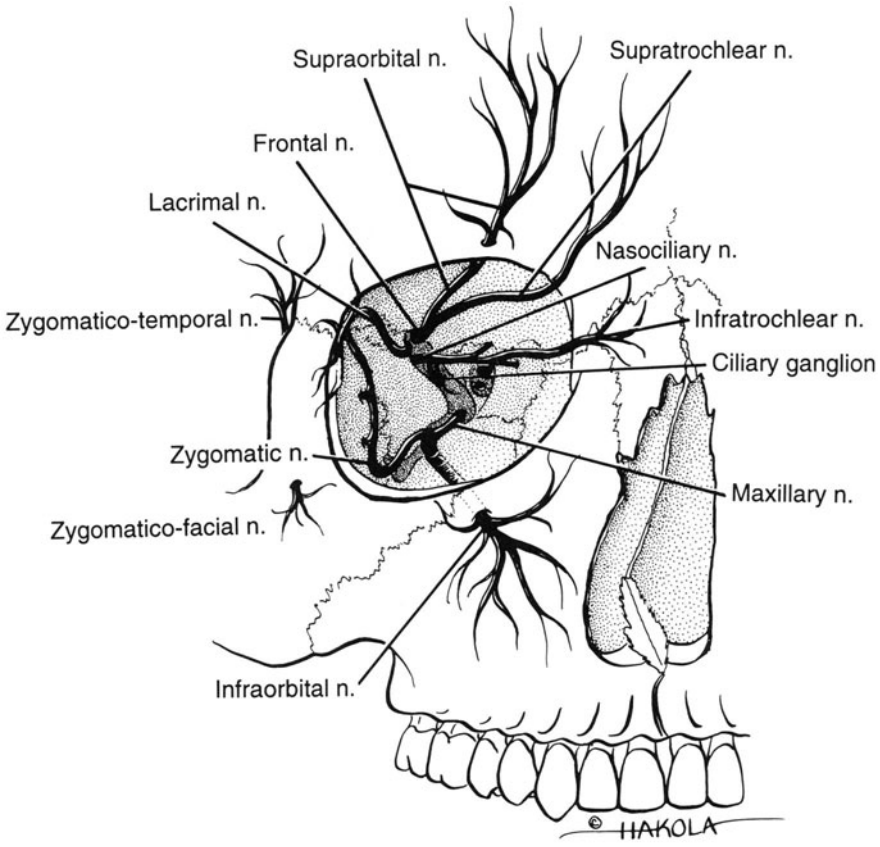

Figure 6.1. Terminal branches opthalmic (v_1) and Maxillary (v_2) divisions of the trigeminal nerve in the orbit.

along the posterior wall of the maxilla. About 20 mm above the maxillary alveolus it enters the tuberosity of the maxilla by one, two, or three branches. These branches are known as the posterior superior alveolar nerves, which supplies the third molar, second molar, and distal half of the first molar. (5) Once the maxillary nerves reaches the orbit in the inferior orbital groove it passes forward, dividing into the middle superior alveolar nerve (present only 60% of the time) and supplies the mesial half of the first molar and the first and second bicuspid teeth. (6) It now enters the inferior orbital canal and here it gives off the anterior superior alveolar nerve, which penetrates the bone to lie along the lateral aspect of the maxillary sinus to supply the cuspid, lateral and central incisor teeth and gingiva. (7) The remainder of the nerve emerges through the infraorbital foramen as the infraorbital nerve, and supplies the skin between the palpebral fissure and the nostril (Fig. 6.3).

The Mandibular Nerve V_3— Mixed Sensory and Motor

The mixed mandibular nerve is mainly sensory and leaves the cranial cavity through the foramen ovale to pass down into the infratemporal fossa. Here it divides off its motor branches, which supply all the muscles of mastication as well as the sensory long buccal nerve. The long buccal nerve runs down on the lateral surface of the buccinator muscle, which it perforates by numerous branches, to supply the gingiva between the second molar and the second premolar teeth. The mandibular nerve continues and then divides into the following four sensory nerves. (1) The *auriculotemporal nerve*, which lies medial to the neck of the condyle of the mandible, turns sharply up and lateral immediately anterior to the external auditory canal, and innervates the skin of the temple, the external auditory canal, and of part of the auditory concha. (2) The *lingual nerve*, which lies between the ramus of the mandible and the medial pterygoid muscle. At the anterior edge of this muscle it curves forward to form an arc whose convex aspect is directed down and back where it enters the tongue from below to innervate its anterior two-thirds (Fig. 6.4). (3) The *inferior alveolar nerve* runs downward, lying first medially to the lateral pterygoid muscle and then running lateral to the medial pterygoid muscle on the inner aspect of the ramus of the manible. Just prior to the inferior alveolar nerve entering the mandibular foramen it is joined by the chordae tympani (a branch of the 7th cranial nerve), which carries taste to the anterior two thirds of the tongue, and preganglionic parasympathetic fibers to the submandibular ganglion. It now passes

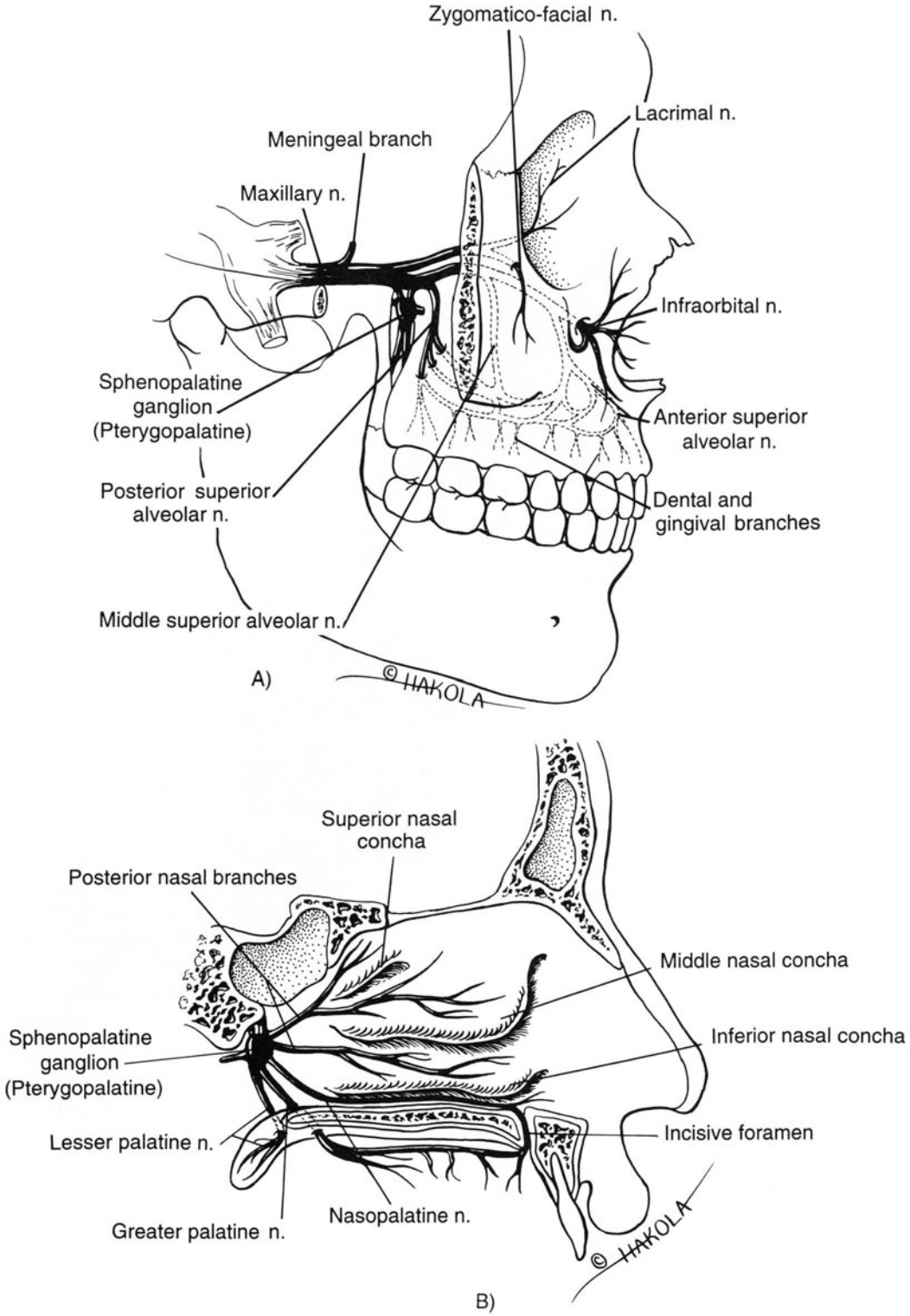

Figure 6.2. (A) Maxillary division (v_2) of the trigeminal nerve. (B) Communicating branches from the maxillary division (v_2) of the trigeminal nerve to the sphenopalatine (pterygopalatine) ganglion.

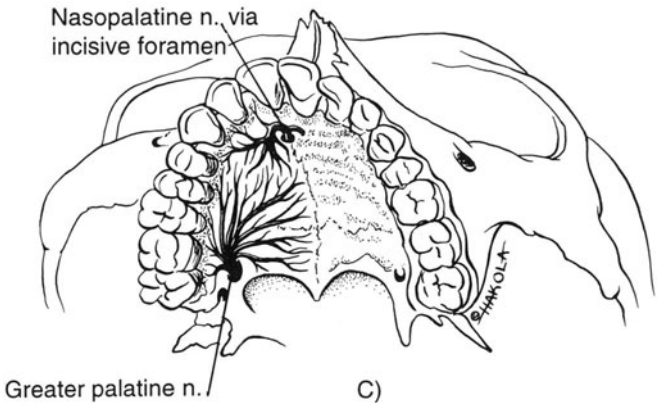

Figure 6.2. *Continued* (C) Terminal branches of the maxillary division (V_2) of the trigeminal nerve that innervates the anterior and posterior palate.

downward immediately behind the lingual nerve and enters the mandibular foramen; as it passes through the inferior alveolar canal, it gives off branches to the teeth and gingiva of the lower jaw. The terminal branch of the mandibular nerve, the mental nerve, exits laterally through the mental foramen and innervates the skin of the lower lip and mandible on each side (Fig. 6.4).

Indications for Intraoral Inferior Alveolar Nerve Block

The intraoral route is most suitable for oral surgery, and restorative dentistry in the lower jaw. Note that the block can be incomplete in the incisor area, owing to the region's bilateral innervation. A contralateral mental nerve block may be necessary to compare the anesthesia of the incisors.

Unilateral operative surgery is possible on the lingual aspect of the alveolar process from the first molar to almost the midline and lateral border of the tongue if the lingual nerve has been blocked as well.

Technique of Inferior Alveolar Nerve Block

The anterior border of the ramus of the mandible, the oblique line, is located with the index finger. A 25-gauge needle is inserted at a point immediately medial to this, and approximately 1 cm above the occlusal surface of the third molar (Fig. 6.5A). The bevel of the needle

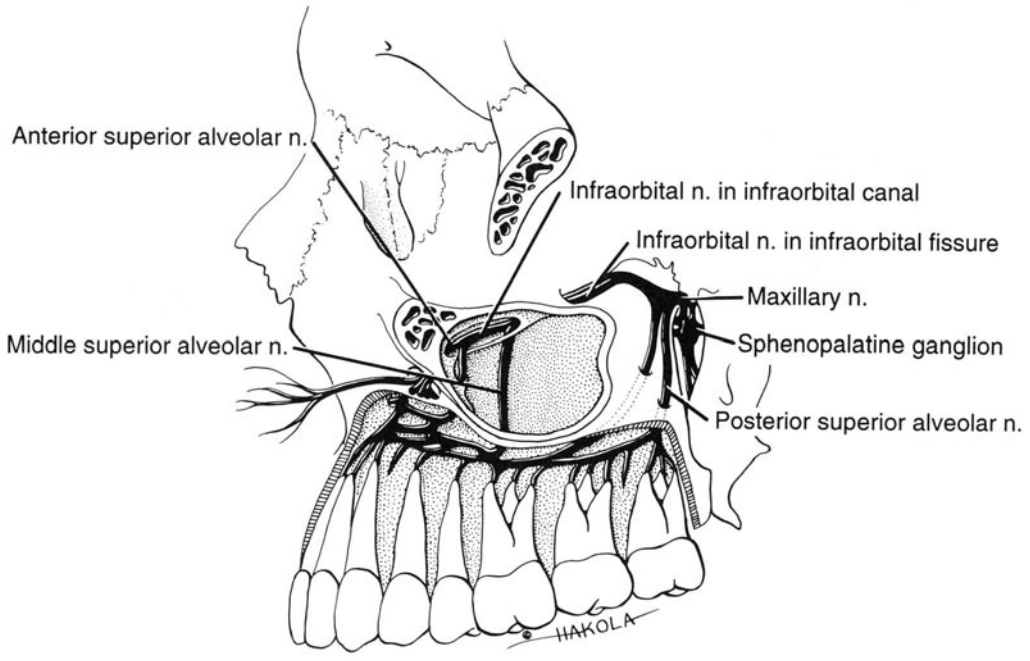

Figure 6.3. Terminal branches of the infraorbital nerve from the maxillary division of the trigeminal nerve to the maxillary dentition.

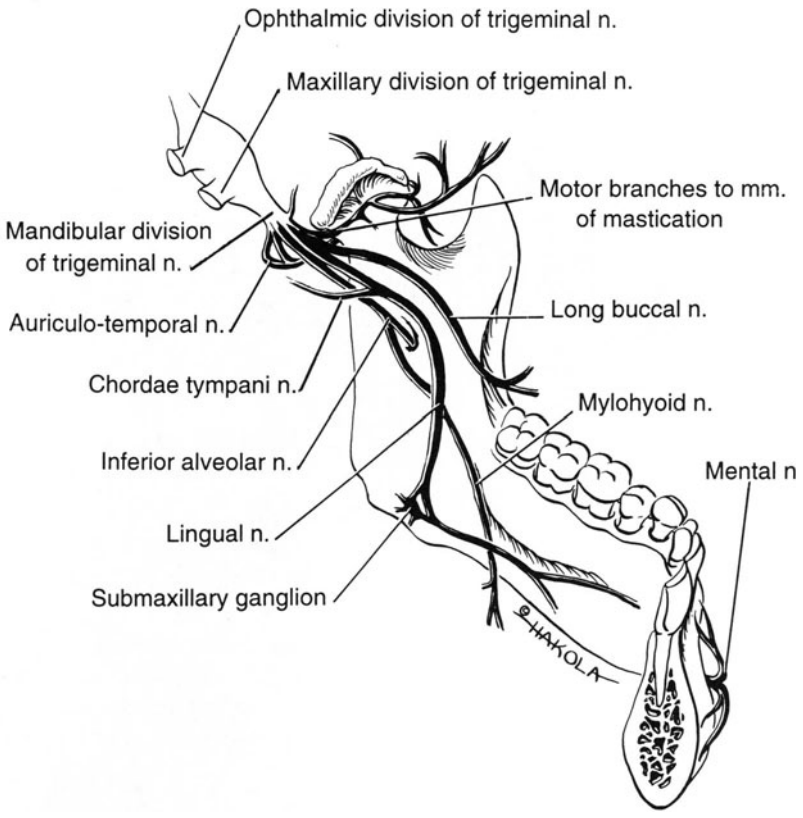

Figure 6.4. Terminal branches of the mandibular division (v_3) of the trigeminal nerve.

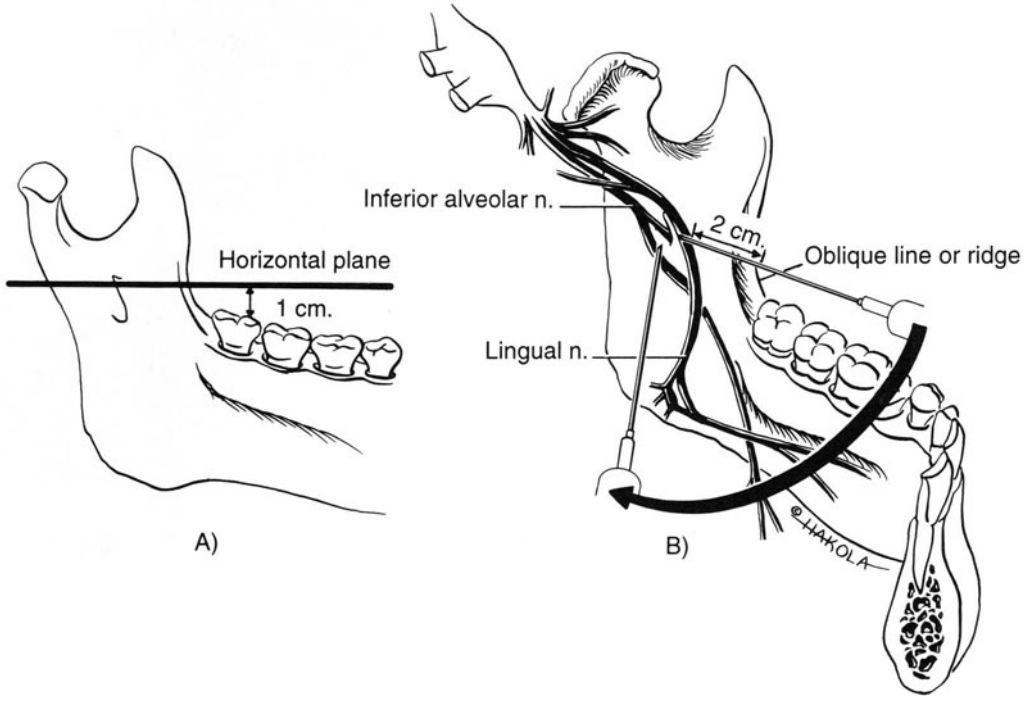

Figure 6.5. (A and B) Technique of inferior alveolar nerve block.

should be directed toward the bone and the syringe must lie virtually parallel to the occlusal surfaces of the teeth of the lower jaw. From this original position, the needle is slowly advanced along the medial side of the ramus to a depth of 2 cm, while swinging the syringe simultaneously over to the contralateral premolar region in the same horizontal plane. The needle must remain in contact with the ramus while this procedure is being performed (Fig. 6.5B).

A higher success rate will be achieved if the patient's mouth remains wide open all the time. If a block of the lingual nerve is required as well, then a small quantity of solution is deposited as the needle passes the oblique ridge. Actually, it is difficult to avoid blocking the lingual nerve, because a small quantity of solution should be injected while the needle is being advanced. When the needle is in the correct position, 1.5–2 ml 2% lidocaine with or without vasoconstrictor should be injected. In edentulous patients, it is especially important to locate the landmarks mentioned and to keep the syringe in the correct horizontal plane.

For extractions in the molar region, the periosteum and mucous membrane on the buccal side of the molars must also be anesthetized because of the long buccal nerve. This may be performed by injecting 0.5–1.0 ml of the anesthetic solution into the buccal mucosa, immediately above its junction with the gingiva opposite the third molar, thus blocking the buccal nerve.

Extraoral Block of the Mandibular Nerve

Indications

Dental and surgical anesthesia is possible in the lower jaw, as well as its mucous membrane and periosteum on both its buccal and lingual aspects and the anterior two thirds of the tongue with the lower part of the cheek. However, general anesthesia is to be preferred when more extensive surgery is to be performed.

This form of anesthesia is suitable for such patients who, because of pain or swelling, cannot open their mouths sufficiently for an intraoral block.

Technique

The needle is inserted into the space formed by the zygomatic arch and immediately anterior to the condyle of the mandible in the sigmoid notch with the mouth open to its widest extent. The needle is advanced at right angles to the skin as far as the base of the infratemporal fossa. The nerve is encountered at a depth of 2–3 cm, lying 1.0–1.5 cm in front of the foramen ovale where 3–4 ml of lidocaine 1–2% with vasoconstrictor are injected (Fig. 6.6).

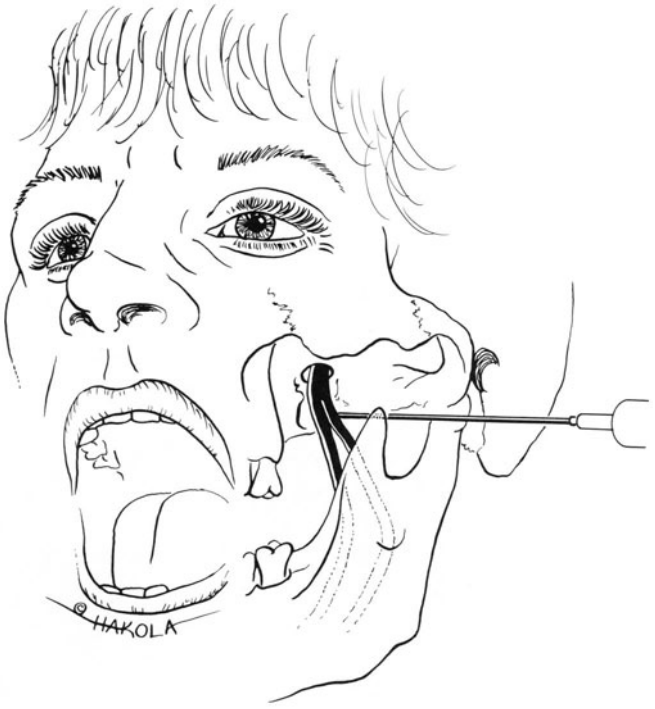

Figure 6.6. Extraoral mandibular block.

Mental Nerve Block

Anatomy

The mental nerve arises in the mandibular canal from the inferior alveolar nerve and exits from the mental foramen on a level with the second premolar. The nerve supplies sensation to the skin and mucous membrane of the lower lip, together with the skin coverage of the anterior mandible and chin.

Technique of Intraoral Mental Nerve Block

The mental foramen is situated at the junction of the lower lip with the attached gingiva just posterior to the first premolar tooth. The index finger is used to palpate the point where the neurovascular bundle exits from the mandible. The needle is inserted from the side until its point is felt in close relation to the neuromuscular bundle, where the boney anterior lingula overlies the mental foramen. Lidocaine 2% (1–2 ml) with or without vasoconstrictor are injected in the area of the neurovascular bundle. Should it not be possible to position the needle exactly over the nerve, adequate anesthesia can often be obtained by depositing the solution around the mental foramen (Fig. 6.7A).

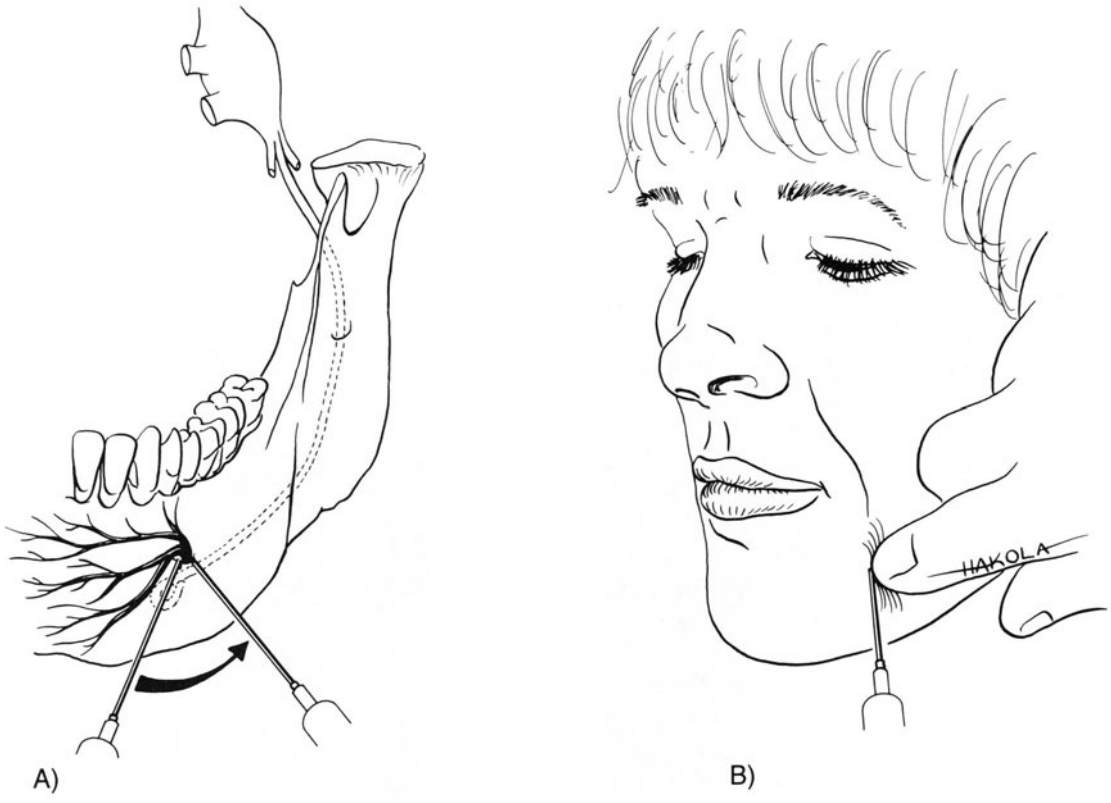

Figure 6.7. Mental nerve block. (A) Intraoral mental block. (B) Extraoral mental block.

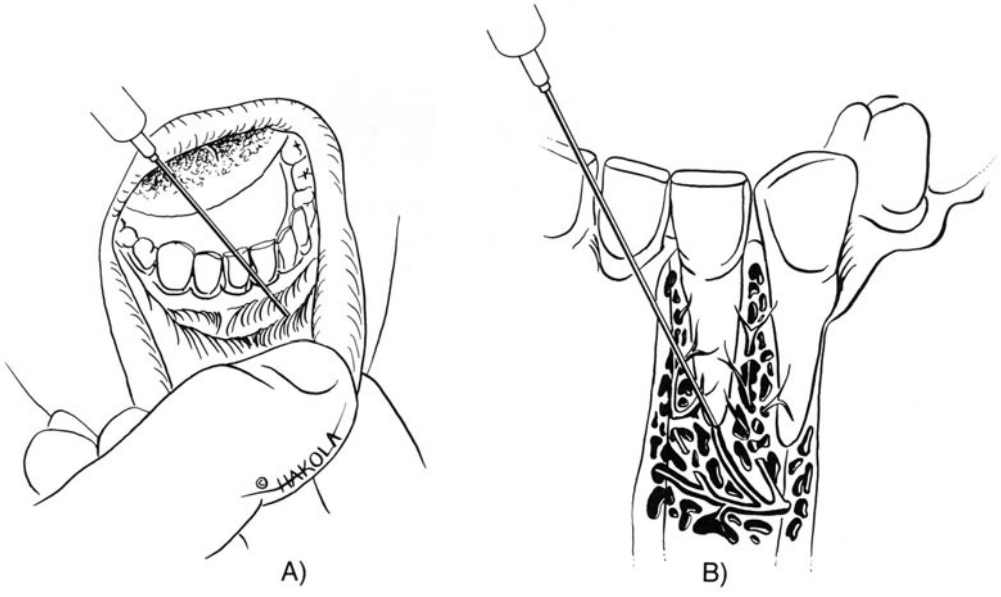

Figure 6.8. Single incisor mandibular block.

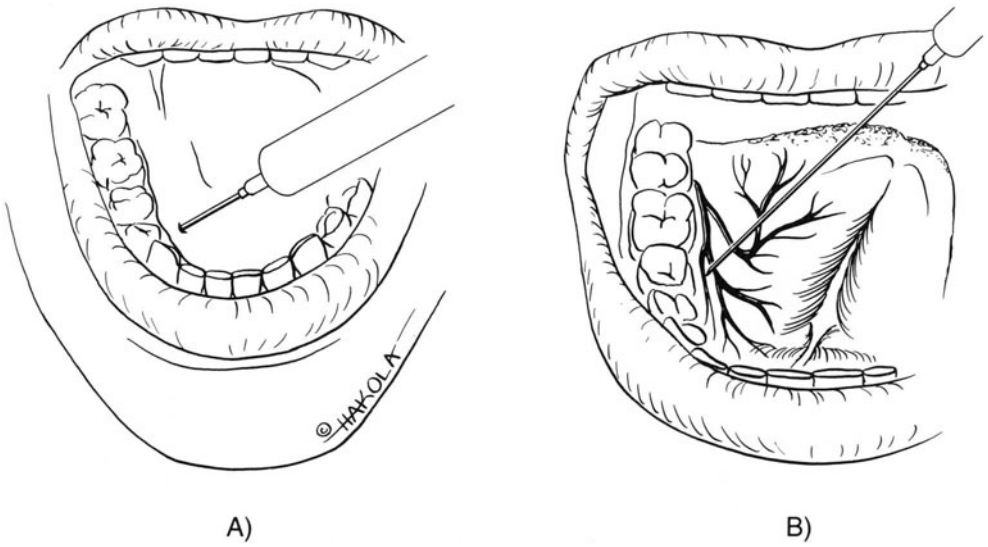

Figure 6.9. Single incisor lingual block.

Technique of Extraoral Mental Nerve Block

The neurovascular bundle exiting from the mental foramen can usually be palpated on the skin surface. Thus, much the same technique is used as for intraoral injection (Fig. 6.7B).

The anesthetized area stretches to the midline of the mandible when the extraoral or the intraoral technique is used. Both techniques may be used unilaterally or bilaterally, depending on the extent of the proposed surgical procedure.

The nerves to a single incisor may also be blocked by submucous infiltration in the sulcus opposite the tooth to be treated (Fig. 6.8A and 6.8B). However, extractions can only be carried out following supplementary anesthesia of the lingual nerve. This is achieved by injecting a small amount of local anesthetic on the lingual aspect, immediately behind the tooth to be extracted (Fig. 6.9A and 6.9B).

Nasopalatine and Greater Palatine Nerve Block

The nasopalatine nerve is the largest of the posterior superior nasal branches. It runs downward and forward along the nasal septum and gives off branches through the incisive canal to the anterior part of the hard palate and the adjacent gingival margin of the upper incisors (Fig. 6.10).

Indications

The intraoral technique is usually employed in dentistry to anesthetize teeth in the upper jaw. For most restorative dentistry, which as a rule only requires analgesia of the pulp, an injection into the buccal fold is sufficient. For surgical interventions, this must be supplemented by a palatal injection for the respective tooth. Extraction of all the teeth of one half of the jaw requires anesthesia of both the greater palatine and the nasopalatine nerves.

The greater palatine nerve supplies the posterior palate and gingiva and is blocked with 3/10 of a mililiter of

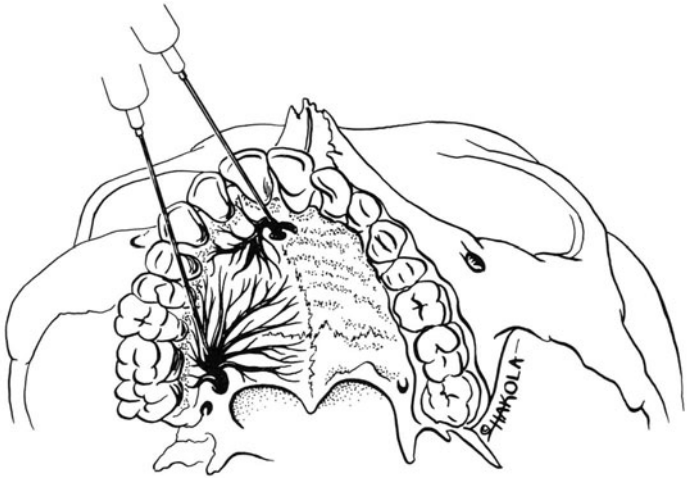

Figure 6.10. Intraoral nasopalatine and greater palatine blocks.

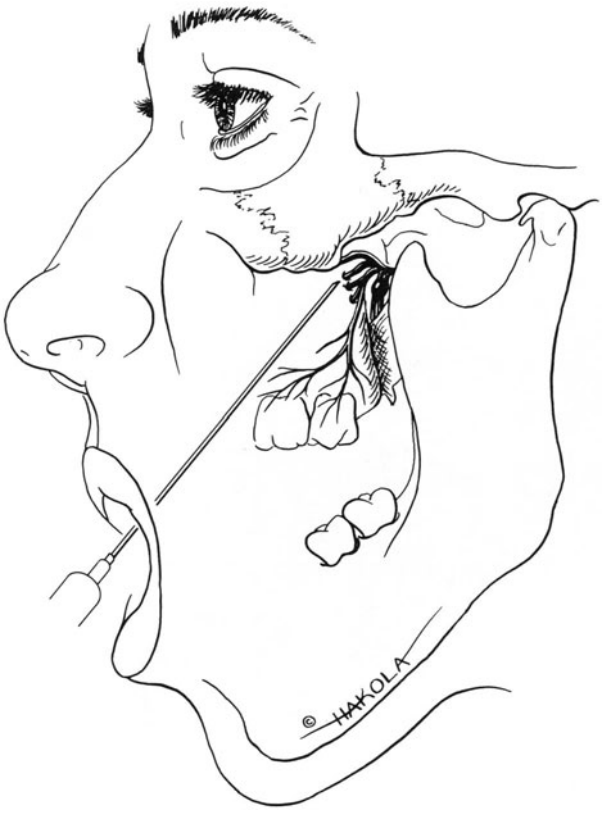

FIGURE 6.11. Intraoral posterior superior alveolar block.

lidocaine 2% with or without vasoconstrictor, in or immediately adjacent to the greater palatine foramen, which may be found at the level of the second molar, 1 cm above the gingival margin (Fig. 6.10).

The nasopalatine nerve is blocked with a few tenths of a mililiter of lidocaine 2% with or without a vasoconstrictor, in or immediately adjacent to the incisive canal, which reaches the middle line immediately behind the incisors (Fig. 6.10).

Posterior Superior Alveolar and Maxillary Nerve Block

The intraoral posterior superior alveolar nerve block is shown in Figure 6.11. The delivery of anesthetic solutions to the middle and anterior alveolar nerves is shown in Figure 6.12A and 6.12B.

Extraoral Technique

The site of insertion is the point of intersection of the lower border of the zygoma and the anterior border of the mandibular ramus. The needle is inserted slightly upward and backward as far as the maxillary tuberosity. The needle is kept in contact with the tuberosity and is inserted further upward until the needle leaves the convexity of the tuberosity and stops when contact with the greater wing of the sphenoid has been made. Here approximately 4 ml of 2% xylocaine, with or without vasoconstrictor, are injected.

Indications

Surgical interventions involving the unilateral skin of the side of the nose, the lower eyelid and the upper lip;

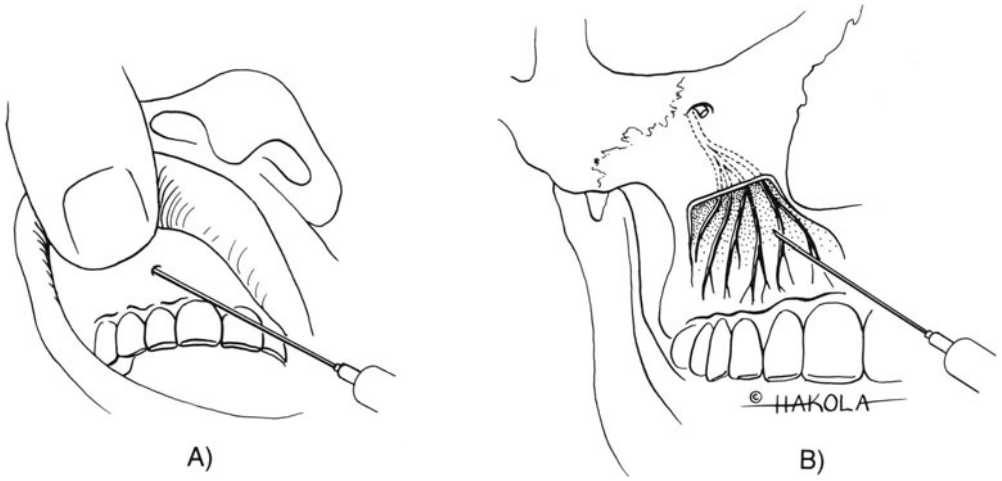

FIGURE 6.12. Intraoral anterior superior alveolar (A) and middle superior alveolar (B) block.

the maxillary bone with the maxillary sinus and the ipsilateral alveolar process, including the ipsilateral teeth, the mucoperiosteum of the palate, and the buccal fold. For more extensive surgery of the maxilla, general anesthesia should be considered.

Infraorbital Nerve Block

Intraoral Route

The midpoint of the infraorbital rim is first palpated by a finger to a point approximately 1 cm below the rim. In

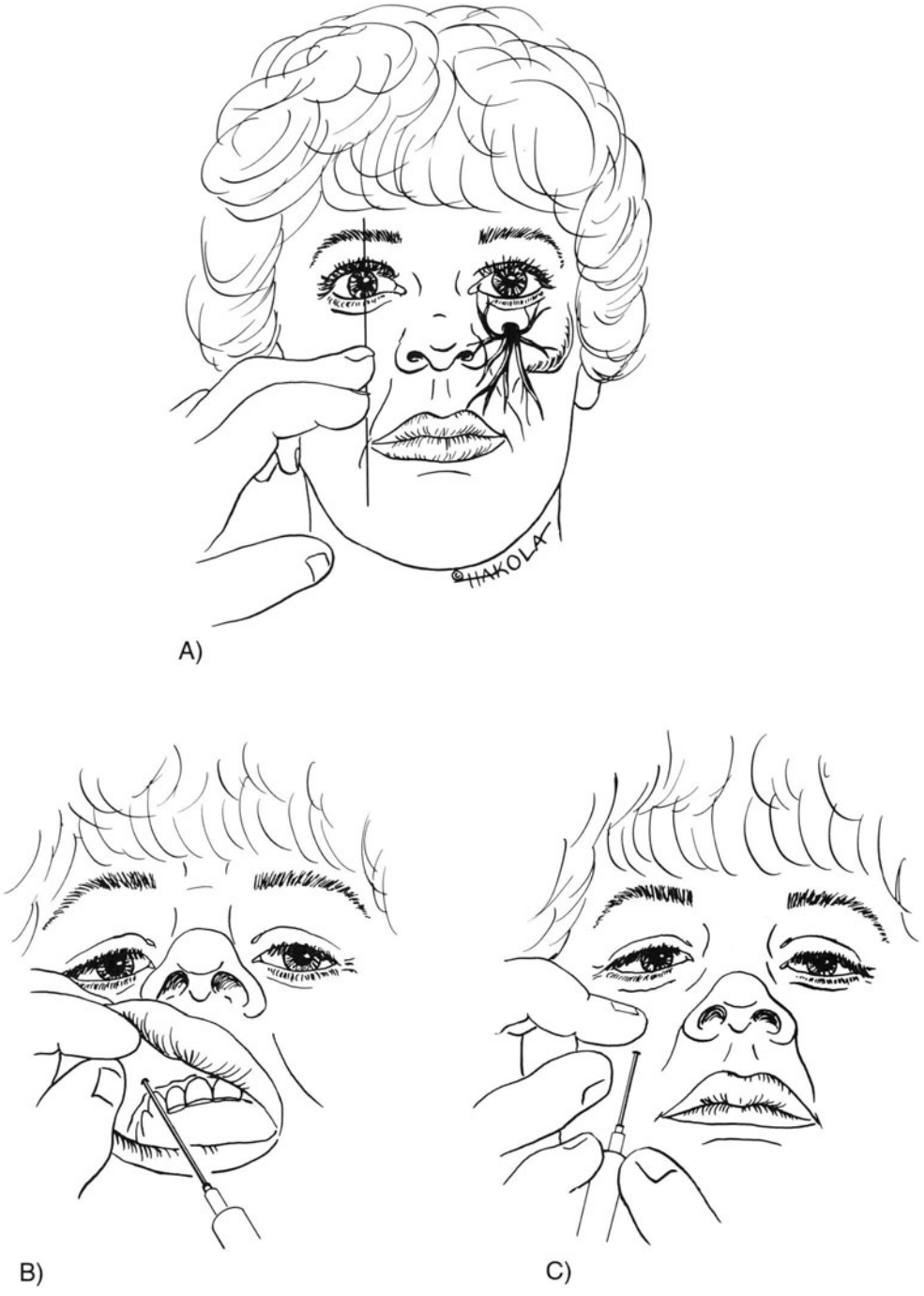

Figure 6.13. Infraorbital nerve block. (A) Extraoral palpation of infraorbital foramen. (B) Intraoral infraorbital block. (C) Extraoral infraorbital block.

most patients, the neurovascular bundle exits the infraorbital foramen at this point and lies on a plane with the pupil of the eye and the corner of the mouth. The needle is inserted into the mucosal sulcus until its point comes to lie under the tip of the palpating finger. The swelling of the tissues as 2–3 cc of local anesthetic solution is injected can be felt by the finger tip (Fig. 6.13A and 6.13B).

Extraoral Route

The needle is inserted into the skin approximately 1 cm below the midpoint of the infraorbital rim and is directed slowly toward the infraorbital foramen. Paresthesia in the area of distribution of the nerve is elicited as the anesthetic solution is deposited in the area of the infraorbital foramen and nerve. Aspiration is attempted to avoid injection into the neighboring artery and vein (Fig. 6.13C).

CHAPTER 7

Basic Principles of Bone Fixation

Bahman Guyuron and Henry C. Vasconez

General Aspects of Bone Healing in the Craniomaxillofacial Skeleton

The skeletal system develops from the mesoderm in utero, which in turn generates mesodermal cells that become organized into embryonic connective tissue known as mesenchyme. Mesenchymal cells, in turn, have the ability to differentiate into fibroblasts, chondroblasts, and osteoblasts giving forth various specific tissue elements. Bone always develops from preexisting connective tissue. The two sources of connective tissue from which bone is formed are mesenchyme and cartilage. The craniomaxillofacial skeleton proceeds from these two types of connective tissue in different sites of its development. The cranial base is predominately of cartilaginous origin; the neurocranium, which will later form the protecting calvaria, and the viscerocranium, which will form the main skeleton of the jaws, are derived from mesenchymal or membranous tissue.

Healing of craniomaxillofacial skeleton fractures occurs in a manner similar to embryonic development. The majority of fractures treated in this area will involve the bone, which is derived of a membranous origin. There will not be an intermediate, cartilaginous, soft callus formation, but rather a type of primary bone healing, which simulates the intrauterine process. Osteoprogenitor cells differentiate into osteoblasts and locally derived osteoclasts direct the fracture healing process. A fibrin structure or matrix is laid down, which then allows for deposition of osteoid tissue. A series of mitogenic inductive growth factors stimulate this process. These factors tend to increase DNA systhesis and cell replication that will lead to fracture healing.

There are many factors that can delay or actually impede normal bone healing. Certain drugs (such as steroids and high dosages of Vitamin A or D), infection, or radiation can all cause delayed fracture healing, or even nonunion. Local factors such as the degree of severity of the injury, the methods of immobilization, and the vascularity of the affected site all play critical roles in fracture healing. The use of rigid fixation in the craniomaxillofacial skeleton came about after a sizable experience in long bone fracture treatment. Rigid fixation has permitted better stabilization of fractures and overall faster and more complete osteosynthesis. Although the bones of the craniomaxillofacial skeleton are, on the whole, nonweight bearing, they do have vector forces exerted on them, especially in the lower jaw region. The process of modeling and remodeling does occur due to overlying soft tissue, tension forces, and muscle pull. Due to the architectural structure of membranous bone, resorption after fractures, particularly in the case of bone grafts, occurs to a lesser extent than with endochondral bone.

Systems of Fixation

Although some of the following fixation techniques might have only historical importance, the maxillofacial surgeon should have the capability and knowledge of fixing the fractured maxillary or mandibular segments under the most adverse circumstances with minimal tools and appliances. For this purpose, a vast array of available techniques for interdental and intermaxillary fixation will be reviewed.

Interdental Wiring

Horizontal or lateral interdental wiring is the simplest approach to approximate the teeth and therefore, the

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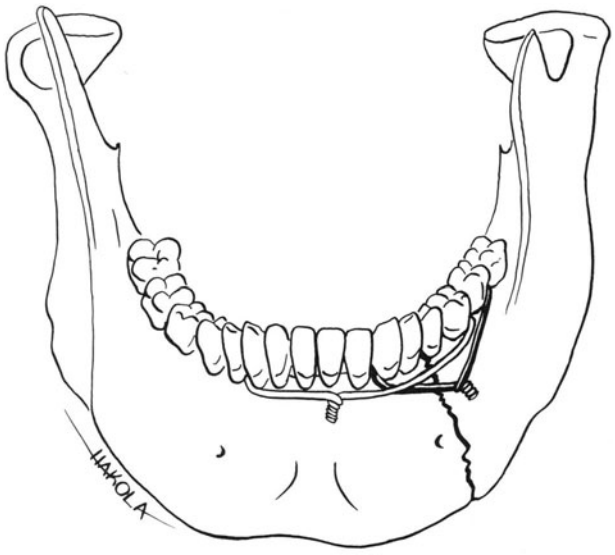

FIGURE 7.1. Wires are passed through the necks of the teeth and twisted together to approximate the fracture.

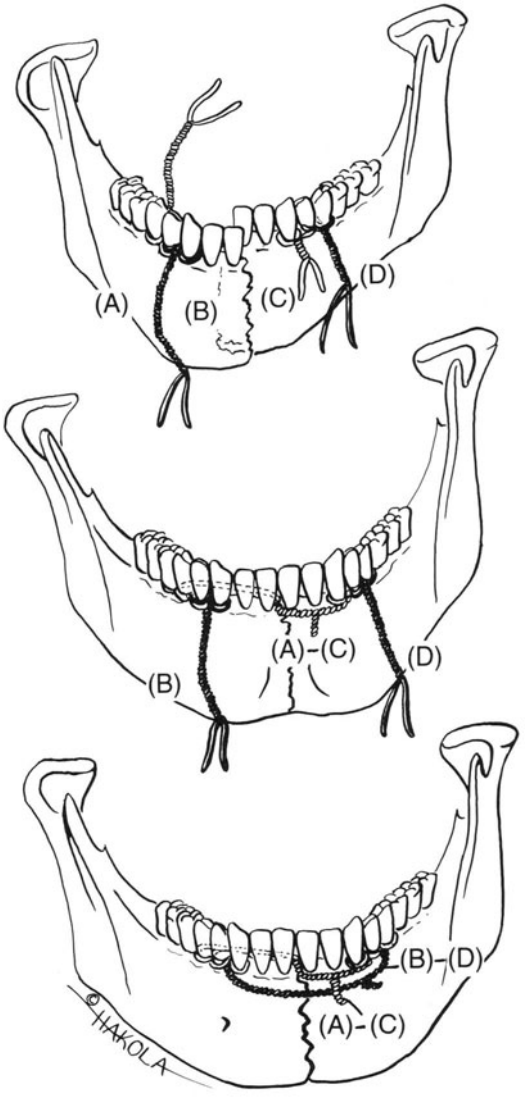

FIGURE 7.2. Wires are passed in a figure-eight fashion with the crossing portion lying between the fractured segments to avoid overriding the segments.

fractured segments. This, obviously, is only possible when an adequate number of teeth are present. It is also critical to be aware that this method only can be used when the teeth are healthy; otherwise, periodontally diseased teeth can be pulled out of the socket somewhat easily.

A loop of #26 or #28 stainless steel wires or #23 gauge brass wires is simply passed around the neck of the teeth and twisted together across the fracture (Fig. 7.1). Another approach is passing the wire in a figure-eight fashion, the crossing portion corresponding to the space in between the fractured segments, which might avoid overriding the segments (Fig. 7.2). It is not safe to use only the teeth immediately adjacent to the fracture line, because it is likely that these teeth will be dislodged. Rather, one should incorporate two to three teeth on either side.

Gilmer Fixation Technique

In 1877, Gilmer used surgical ligatures to establish intermaxillary fixation by taking the wires across the occlusal plane, having ligated them around the upper and lower incisors[1] (Fig. 7.3).

Another technique, described by Gilmer in 1881, is the circummandibular wire, which is particularly suitable for patients with fractures of the mandible.[2]

Cable Arch Wires

A large-sized wire, such as a 0.02 brass wire or #24 gauge stainless steel wire, is passed around the neck of the molar tooth on one side and then twisted firmly, leaving a long strand. This is used as a stand and pivot point for the rest of the dental arch extending to the opposite molar (Fig. 7.4). Finer wires, such as 0.03 gauge brass or 26 or 28 gauge stainless steel, are similarly ligated around the other teeth successively and twisted around the main anchor wire. The end of the finer wire is cut and bent toward the teeth to avoid discomfort to the patient. This procedure is continued step-by-step until the last molar on the opposite side is ligated in the arch.

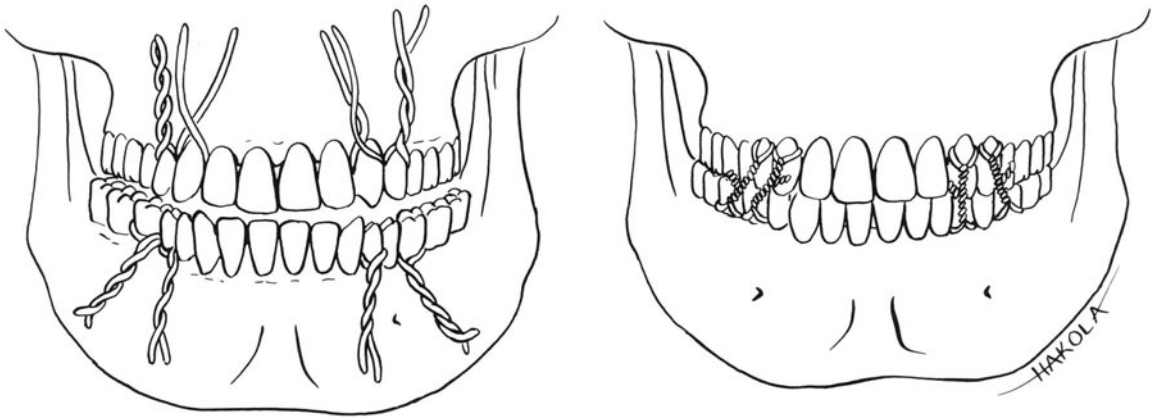

Figure 7.3. Gilmer's method of passing wires across the occlusal plane (Left), then ligating them to each other (Right).

Ivy Loop

In 1922, Ivy described an easy and effective technique of interdental wiring. A #22 or #24 gauge stainless steel wire is selected with an approximate length of 4–6 inches (Fig. 7.5A). It is bent in the midportion and an eyelet is created using a hemostat or a fine wire twister. Both ends of the eyelet wire are inserted through the interdental space from the labial to the lingual aspect. The two ends are then separated and each one is passed in between adjacent teeth. The ends are then twisted together. These free ends can either be passed through the eyelet or, even better, under the loop adjacent to the teeth. After the free ends have been ligated, they are then cut and bent. Having ligated an adequate number of teeth using this method, the eyelets are then bent cephalad on the maxillary arch and caudally on the mandibular arch as an anchor (Fig. 7.5B). The maxillary and mandibular arches are then wired together by passing several wires from maxillary eyelets to the mandibular wires, while a normal occlusion in stabilized. These anchors can also be used for the suspension of rubber bands.

Dental Arch Wire Appliances

Based on Angle's design, introduced in 1890, ready-made bands that encircle the crowns of the teeth have been used successfully for many years. A nut, jackscrew, and wire soldered to the band comprise the fixation device (Fig. 7.6). The bar is made of 14 gauge brass wire flattened to 19 gauge. The rationale behind the use of brass is its quality of malleability, so the bar can be adapted to the contour of the mandibular or maxillary arch. This bar is then ligated to the teeth using finer wires. Additional spurs can be soldered to serve as the anchor for the wires or elastic bands.

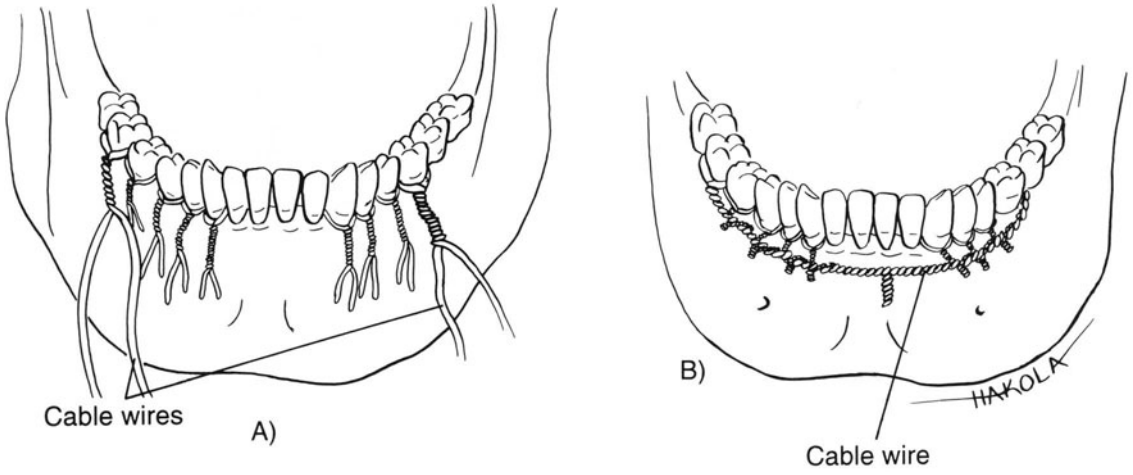

Figure 7.4. With the cable arch wire approach, a heavy wire is ligated on one side of the arch with finer wires twisted around the remaining teeth (A). The heavier wire is twisted along with each finer wire to create a cable arch wire (B).

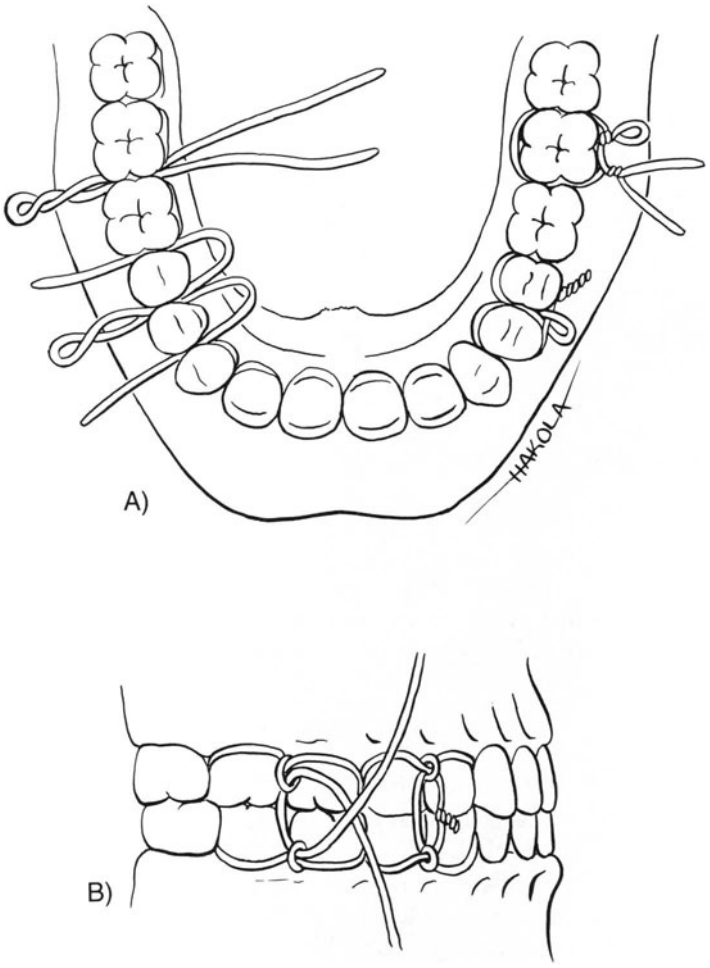

FIGURE 7.5. The Ivy Loop method of creating eyelets for interdental wiring (A). After bending the eyelets in the appropriate direction, the maxillary and mandibular arches are wired together (B).

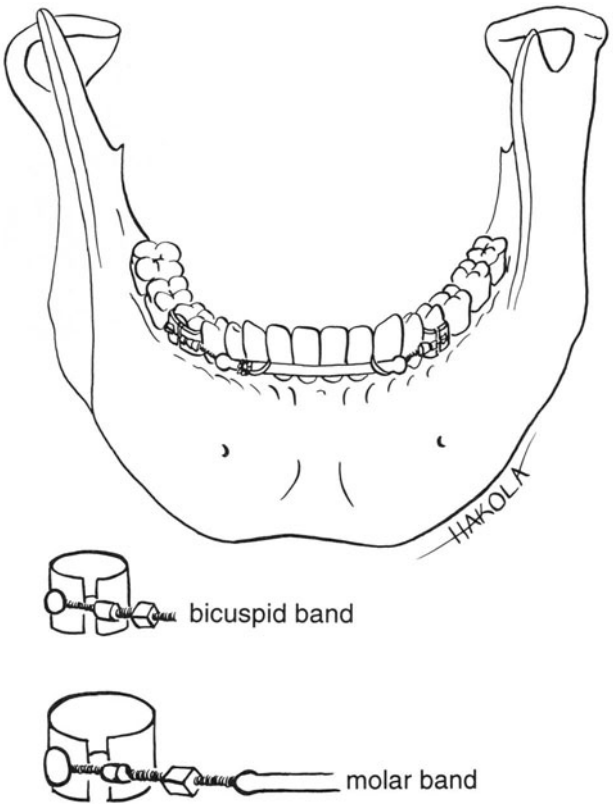

FIGURE 7.6. A dental arch wire appliance consists of a nut, jackscrew, and wire soldered to the band.

The Continuous Stout-Interproximal Wire Loop

This technique, which has been used since 1942, can only be used when at least three adjacent teeth are present and healthy. This technique is useful in creating suspension hooks for elastic bands. To construct successive buccal surface loops, the wire is passed through the interdental space, preferably between the second and third molars. The buccal portion of the wire is placed against the gingival margins of the teeth that have been chosen for wiring, and a bar 3 mm in diameter is placed next to the tooth (Fig. 7.7A and 7.7B). The lingual portion of the wire is passed through each interdental space on the buccal surface, in turn, forming a loop over the bar. The wire is then passed lingually through the same interdental space. Subsequent to the placement of an adequate number of loops, the end of the wire is loosely twisted in either the cuspid or bicuspid region (Fig. 7.7C–7.7F). Prior to the final tightening, the bar is removed, which will leave a series of loops on the buccal side of the teeth. Each loop is twisted a few times, so it can be used as a hook. Should a tooth be missing, the buccal and the lingual arms of the wires are twisted to bridge the space. The free ends of the loosely twisted wires are now tightened adequately.

Kazanjian Button

This method is used to create anchors for elastic bands during intermaxillary fixation. Two pieces of wire are passed around each of the two adjacent teeth, making four free ends available on the labial side (Fig. 7.8A). These are then ligated around each of the two teeth. The ends of the two twisted wires are then twisted together and trimmed to a 1-inch length (Fig. 7.8B). This is then bent a few times to take the shape of a button (Fig. 7.8C and 7.8D). Bicuspids or molars are more suitable for this method. These buttons can be used to fix simple fractures without significant displacement of the fragments, serving as an anchor for the intermaxillary wires or elastic bands.

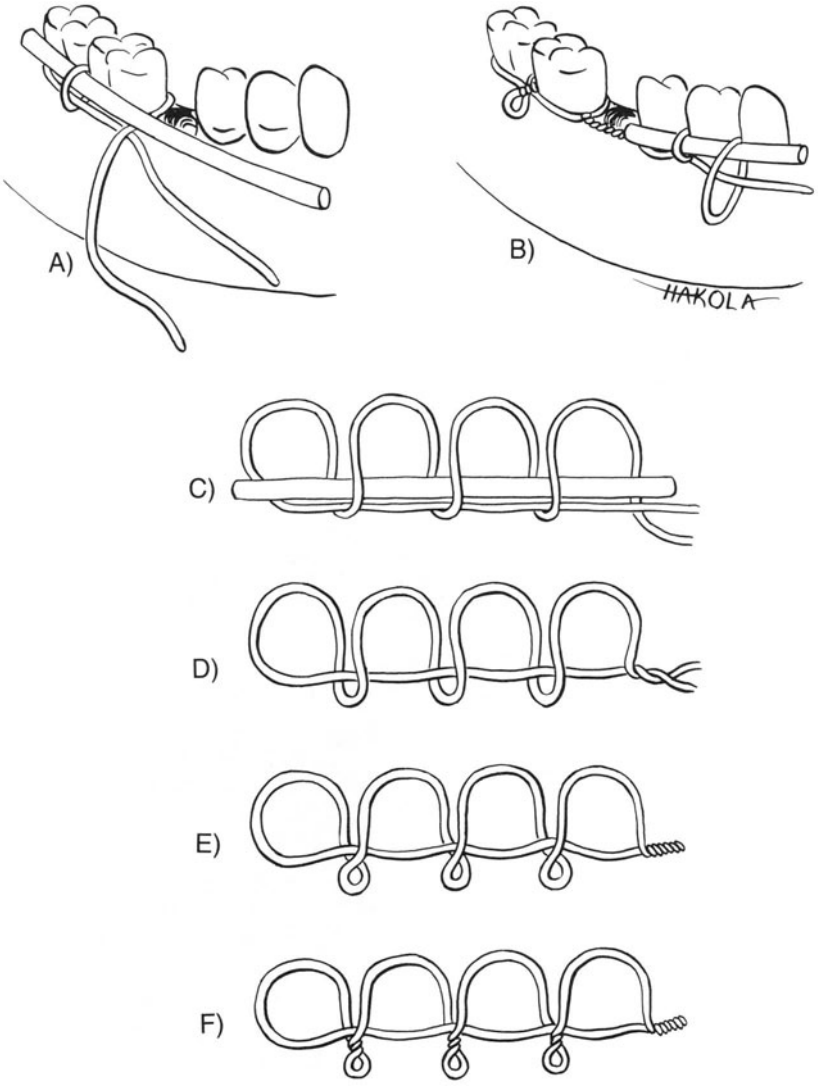

Figure 7.7. With a Continuous Stout-Interproximal technique, loops are created over a 3-mm bar next to the labial side (A and B). The loops are twisted to take the shape of a hook (C, D, E, and F).

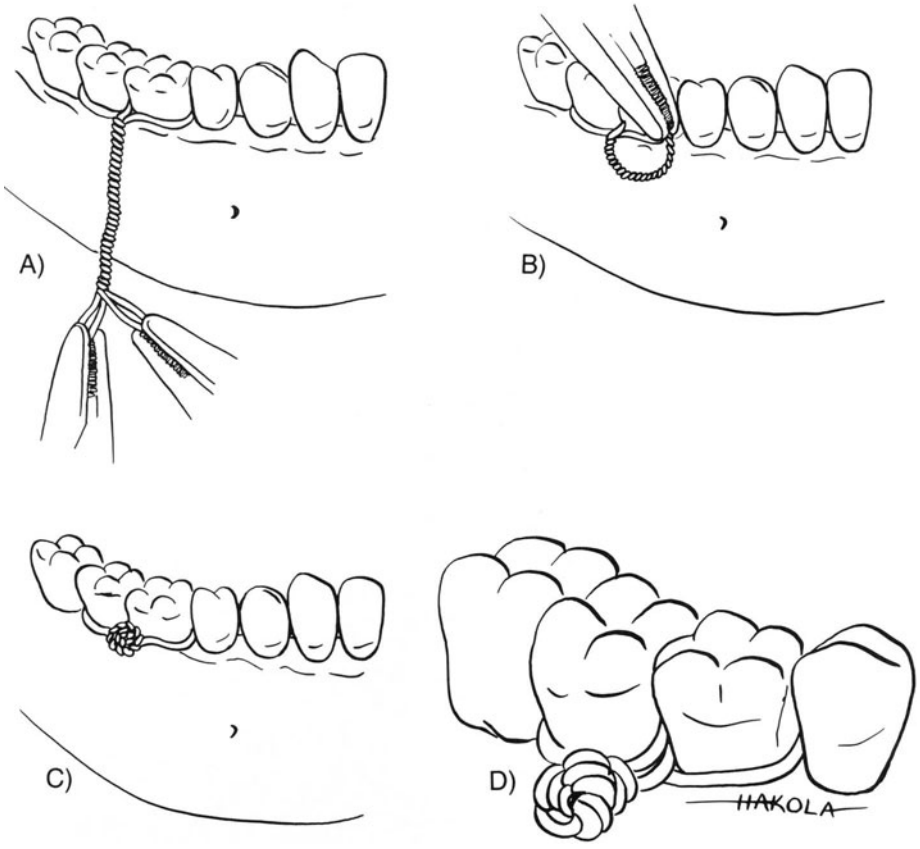

Figure 7.8. To form a Kazanjian button, two pieces of wire are passed around two adjacent teeth and twisted together (A). The ends are twisted and trimmed to a 1-inch length (B). These wires are bent into the shape of a button (C and D).

Orthodontic Appliances

Although not proper for emergency conditions, ordinary orthodontic appliances with rectangular wires provide the most satisfactory means of intermaxillary wiring. These are applied selectively and neatly, do not endanger the periodontal tissue, and can provide adequate stability, especially if the bands are used around more stable posterior teeth.

Erich Arch Bars

Winter or Jelenko type arch bars, modified by Erich, provide the most practical intermaxillary wiring in an emergency situation. They are rarely used for elective orthognathic surgery in patients who have already had previous orthodontic alignment of the teeth. However, the orthodontic appliances are far superior to Erich bars in elective orthognathic surgery. There are prefabricated wire bars that have special extensions for use as anchors. The bar can be ligated to the teeth using #26 or #28 gauge stainless steel wire (Fig. 7.9). The ends of the wires are then bent toward the teeth to avoid irritation of the mucosa on the buccal surface.

Sectional Appliances

In practice, this type of appliance does not provide ideal stabilization (Fig. 7.10A–F). However, when there are multiple fractured segments with displacement, each alveolar segment can be attached individually to a bar using wire ligatures around the tooth. The segments can be attached to each other ligating the bars together with the ideal arch alignment of the segments. With the availability of rigid fixation systems, however, this type of fixation is even less likely to be used. Nevertheless, this should be part of the armamentarium of maxillofacial fixation, in case the rigid fixation system or tools are not available or they are deemed unsuitable.

Cap Splints

Cap splints are mostly used overseas and have not gained popularity in the United States. Designed to cover the occlusal and exposed portions of the teeth, cast caps require the services of skilled dentists or dental technicians. These, however, provide strong fixation appliances. Because the cast caps are cemented to the occlusal surface, the exact occlusion cannot be ascertained with the splint in position. This often results in occlusal imperfections, which will have to be corrected orthodontically or by occlusal adjustments subsequent to removal of the splint. Therefore, transparent acrylic cap splints, which provide some visibility and more precision, are preferred.

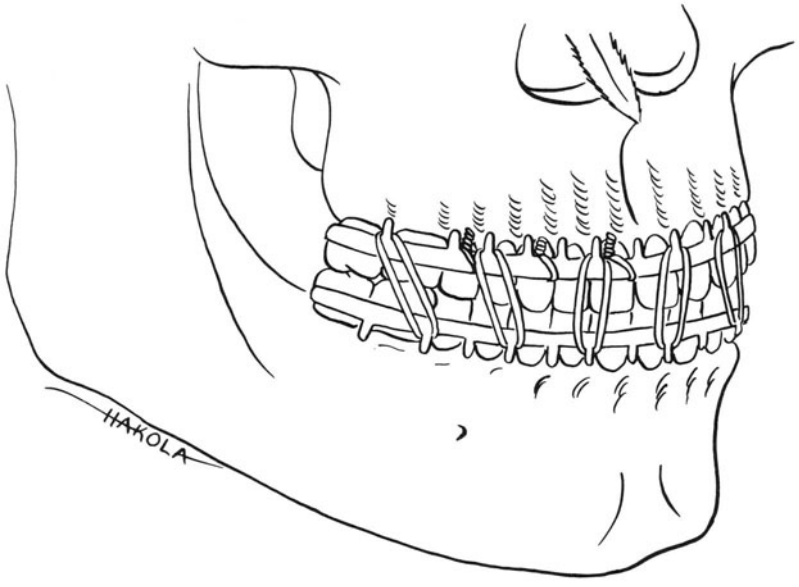

Figure 7.9. Erich arch bar—Winter or Jelenko type arch bar, which is ligated to the teeth, has prongs for further fixation with wires or elastic bands.

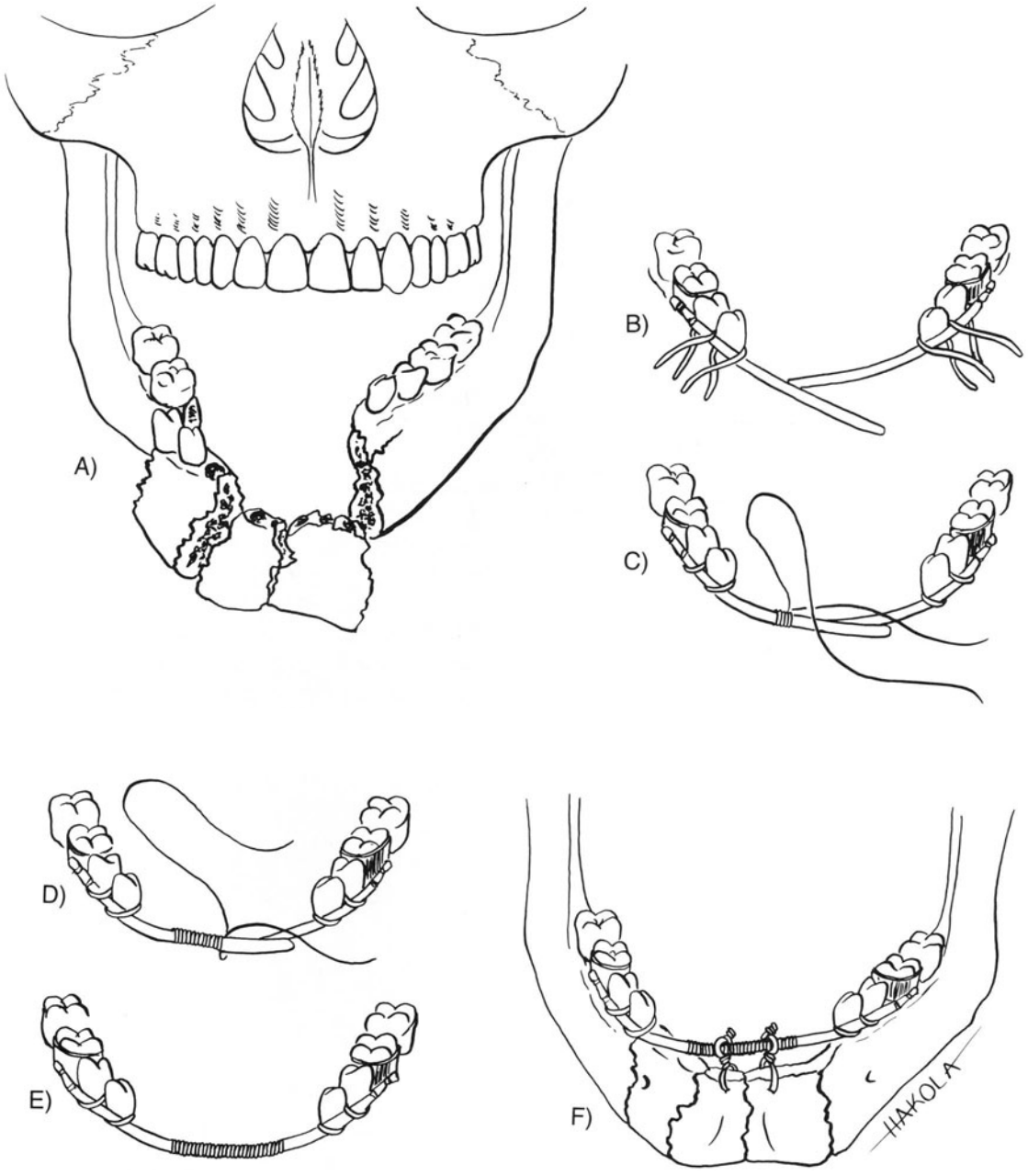

Figure 7.10. Type of fracture that is suitably fixed with a sectional arch wire (A). An arch wire splint is banded to the first molar on each side and then wired to the other teeth (B). After manipulation of the fracture sites, the arch wires are ligated together (C, D, and E). The floating segments are suspended from an arch wire (F).

Definitive Banded Retention Appliances

The most precise approach to alignment and fixation is grouping of the teeth and application of segmental bars, bands, and wires. These bars can then be fixed on top of each other by a splint, which has to be made in the laboratory. This method requires a thorough knowledge of anatomy and occlusion. A committed laboratory technician, orthodontist, or surgeon knowledgeable in this area can fabricate these splints and fixation devices (Fig. 7.11A–F). However, this technique is seldom applicable in the emergency setting.

Interosseous Wires

Although Buck was the first to place an interosseous wire in the mandibular fracture in 1847, Adams is the one who popularized this practice (Fig. 7.12).[3,4] He used a combination of wires for the fixation of fractures, including interosseous wires, which were supported with suspension wires extending from the zygoma to the maxilla for maxillomandibular fixation (MMF).

Intermedullary Pins

Intermedullary pinning was first advocated by Major in 1938, then widely used by McDowell, Barrett-Brown, and Fryer for maxillofacial fractures (Fig. 7.13).[5,6] During world War II, pin fixation became popular. Anderson's orthopedic external pin fixation was expanded to mandibular fractures by Converse.[7]

Metallic Mesh Implants

The use of metallic mesh implants made of stainless steel, chrome-cobalt, or titanium have been popular choices for mandibular surgery since 1967 (Fig. 7.14).[8–12] The metallic mesh was initially used for reconstructive procedures such as malunion. Its use was later expanded to maxillomandibular fractures.

Bone Clamps

In 1972, bone clamps were introduced, which function similar to a vise, as these devices were clamped to the buccal and lingual cortices under the inferior border of the fracture site.[13]

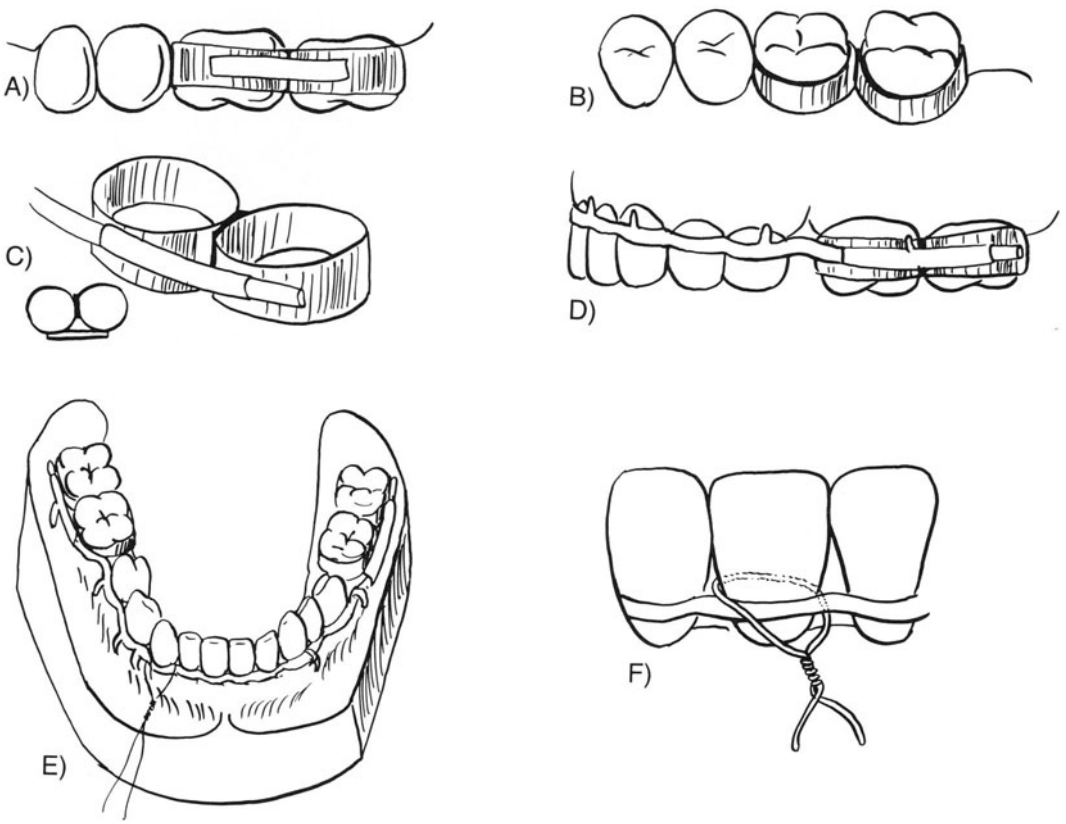

Figure 7.11. Orthodontic bands are fitted onto two adjacent teeth (A and B). The bands are soldered together and a horizontal tube is soldered to the buccal surface (C). An arch bar is attached to the teeth by slipping the ends through the buccal tubes and wiring the bar to the teeth (D). An example of a banded retention appliance on a plaster cast (E). The arch wire is further secured by ligating it to the teeth with wire (F).

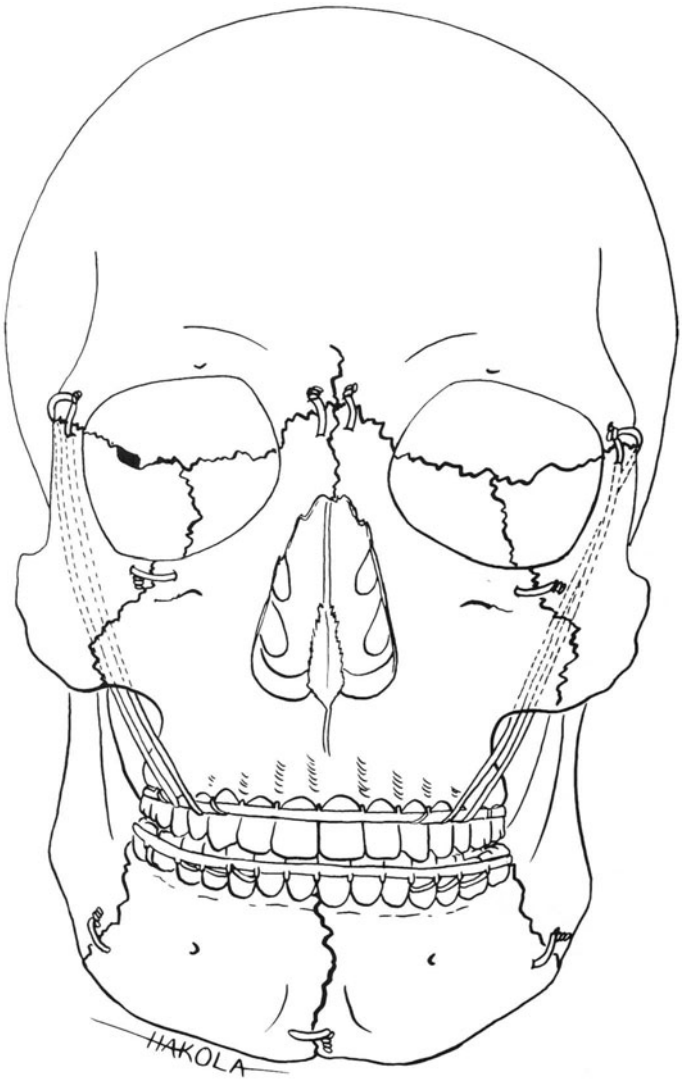

FIGURE 7.12. Adams' method of interosseous and suspension wires to treat maxillofacial fractures.

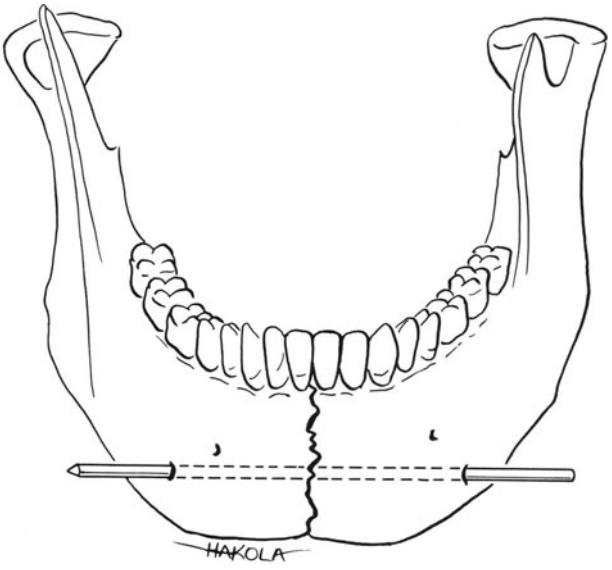

FIGURE 7.13. Intermedullary pinning to stabilize a mandibular fracture.

1. The treatment should be conducted in such a manner that the fracture and soft tissue surrounding it are ensured thorough asepsis.
2. Any implant should not be chemically or electrically active or cause mechanical irritation.
3. Internal fixation should create and maintain absolute interfragmentary rigidity until natural bone union has occurred.
4. Internal fixation should create and maintain absolute interfragmentary pressure at the fracture site, mainly directed along its axis.[17]

Rigid Fixation

A practical use of the plate was first described in 1886 by Hansmann.[14] He used a nickel-plated steel strip with screw holes drilled at various intervals with the screws protruding through the wound. Although Lane developed a steel plate in 1893 that bears his name, his biggest contribution to internal rigid fixation was the emphasis he made on aseptic technique.[15] Sherman made a major contribution to the development of fixation devices through his design efforts of screws and plates in collaboration with the Pittsburgh steel companies.[16] The biggest contributions, however, were made by Danis (from Belgium) in the mid-20th century. He established some principles of fixation that are generally accepted today. These include:

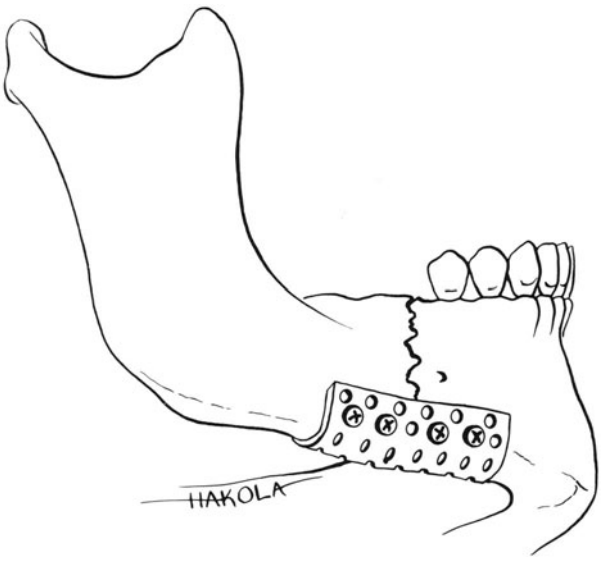

FIGURE 7.14. Metallic mesh plate used to fixate a mandibular fracture.

Bone plates, according to the literature available, were first introduced to maxillofacial surgery by Christiansen in 1945.[18] He used tantalum plates to provide stability in between the fractured mandibular fragments.[19] Coincidentally, Winter and colleagues used a Vitallium prosthesis for mandibular reconstruction the same year.[20]

The Association for Osteosynthesis (AO), which originally convened in the mid-1950s, developed four principles for the treatment of skeletal fractures with the goal of full function:

1. Accurate anatomical reduction.
2. Atraumatic operative technique, which preserves the vitality of the bone and soft tissue.
3. Rigid internal fixation, which produces a mechanically stable skeletal unit.
4. Avoidance of soft tissue damage and "fracture disease" by allowing early, active, pain-free mobilization of the skeletal unit.[21]

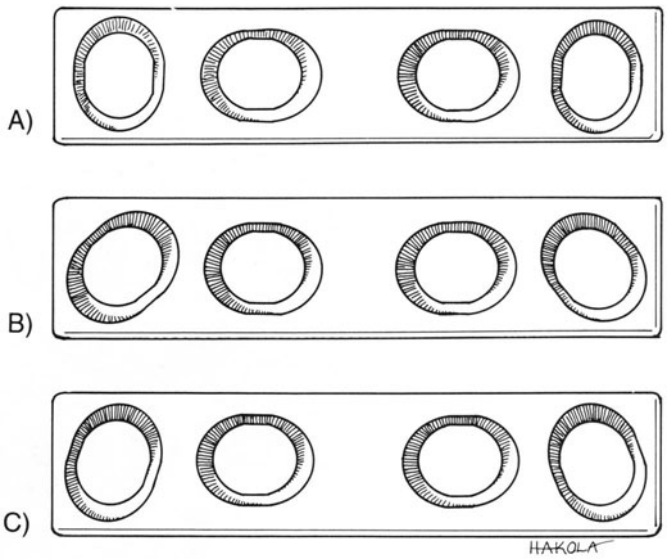

Figure 7.15 Initial attempts on the eccentric dynamic compression plates had the terminal hole in a 90-degree position (A), then at 45 degrees (B), now at a 75-degree position (C).

Further cases of plate and screw fixation were then introduced periodically. In 1958, Bagby and Janes ingeniously designed a compression plate.[22]

Although the 1960s marked the experimental period for plate and screw fixation of maxillofacial fracture, the early 1970s marked the beginning of clinical application. This, along with some well-designed scientific studies, laid the foundation of the knowledge that we have concerning rigid fixation modalities, their proper applications, and an understanding of the mechanical principles underlying rigid fixation.

Most of the rigid internal fixation studies during the 1960s were done by Luhr, who felt the addition of compression across the mandibular fracture would provide the necessary rigidity in between the fragments to eliminate the need for maxillomandibular fixation. In 1968, Luhr reported his work on a mandibular plate, which followed the compression principle.[23] That same year, Mittelmeier also wrote an article on the compression plate.[24]

Besides the introduction of the compression system to maxillofacial surgery, Luhr popularized the self-threading screw, which has been modified over the years. Luhr's most recent contribution to craniomaxillofacial surgery is the Micro-fixation and Panfacial systems, which both have significant clinical applications.[25]

Spiessl also has contributed a great deal to the development of the mandibular system in the 1960s and 1970s through his work with the AO/ASIF. Two versions of the eccentric dynamic compression plate were ingeniously developed in 1973 by Schmoker and Niederdellman.[26] Once placed in the usual location along the inferior aspect of the buccal cortex, this plate provided adequate compression at the alveolar region. Although these two investigators developed their own systems independently, both types produced compression in the alveolar region by modification of the terminal compression holes so they were no longer parallel to the long axis of the plate.[18] These two plates have been replaced with the AO/ASIF eccentric dynamic compression plate with a 75 degree hole (Fig. 7.15).

Another contribution was the introduction of lag screws. This technique can be accomplished by means of a screw with a potential for lag, which means that only the terminal portion of the screw is threaded. The same effect can be attained by overdrilling the burr hole on the segment closer to the screw head. Consequently, there is no potential for threading on the outer cortex, while the segment away from the screw head will be threaded. Therefore, when the screw is tightened, the cortices of the two segments will be approximated. This technique was first introduced to maxillofacial surgery by Brons and Boering in 1970.[27]

The next logical step in the advancement of maxillofacial rigid fixation procedures was the intraoral approach, which diverged from Luhr's and Spiessl's techniques of extraoral rigid fixation (Fig. 7.16).[28–32] In 1973, another advancement was made in the management of maxillomandibular fractures; the introduction of monocortical screws by Michelet.[29]

Just after introducing the Luhr Microplate system in the late 1980s, Professor Luhr created the Panfacial system. The screws and plates used in these systems are slightly larger than those used in the Microsystem, but smaller than those in miniplates. The specific reason for

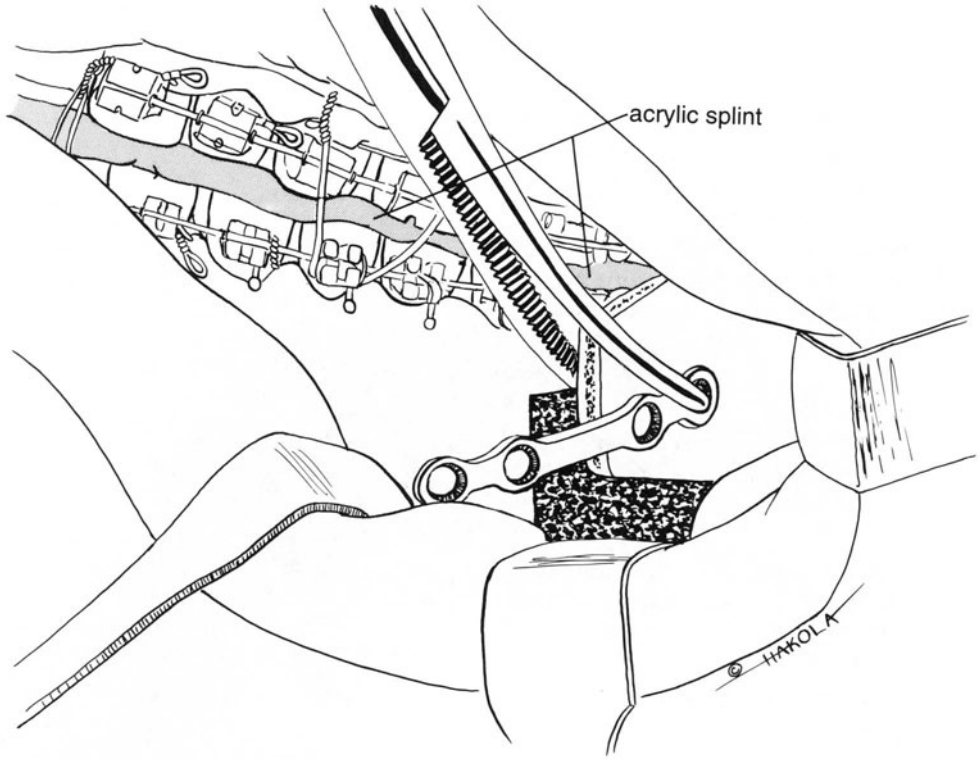

FIGURE 7.16. Intraoral approach to rigid fixation following a sagitally split mandibular osteotomy.

the introduction of this system was the need for low-profile plates that could not be palpated, yet would provide more strength than the Microsystem.

Anatomy of a Screw

A screw has two major anatomical parts—one is the head and the other is the shaft (Fig. 7.17).

Head. This is the larger and wider portion of the screw, which can be cast in a straight slot with a central depression (Wurzburg), Philips (Luhr), cruciate (Synthes), or Hex (Synthes) shape to accommodate several types of screwdrivers.[18] The head has several components; one is the countersink. This is the tapered or beveled undersurface of the head of the screw. The countersink angle is the angle between the beveled inferior margins of the head on either side. Without a coutersink angle, the head will be thick and high profile. The wider the angle and the less thickness of the head, the less profile.

Shaft. The shaft, which is the portion that contains the screw threads, has two diameters. One is the core diameter (the minor diameter), the other is the external diameter (major diameter), which is the diameter including the width of the screw and thread portions. The core diameter corresponds to that of the pilot hole drilled in the bone. The major diameter or external diameter is the actual screw size. For example, a 2 mm screw size has an external diameter of 2 mm.

The portion of the screw immediately below the screw head is called *run-out*. The *pitch* is the vertical distance between the edge of the two consecutive screw threads along the external surface. The difference between the major and minor diameter is called thread depth. The terminal end of the screw is referred to as the point. There are three types of thread configurations (Fig. 7.18):

1. The buttress thread is the most popular thread configuration. The superior portion of the thread, meaning the closest to the screw head, is almost perpendicular to the shaft or core of the screw. When the screw is tightened, compression is primarily provided between the horizontal aspect of the buttress thread and the inferior portion of the screw head (countersink).
2. The v-shaped thread, which is a pointed thread, is the most common design for self-tapping screws. The superior and inferior ends of each thread are angled

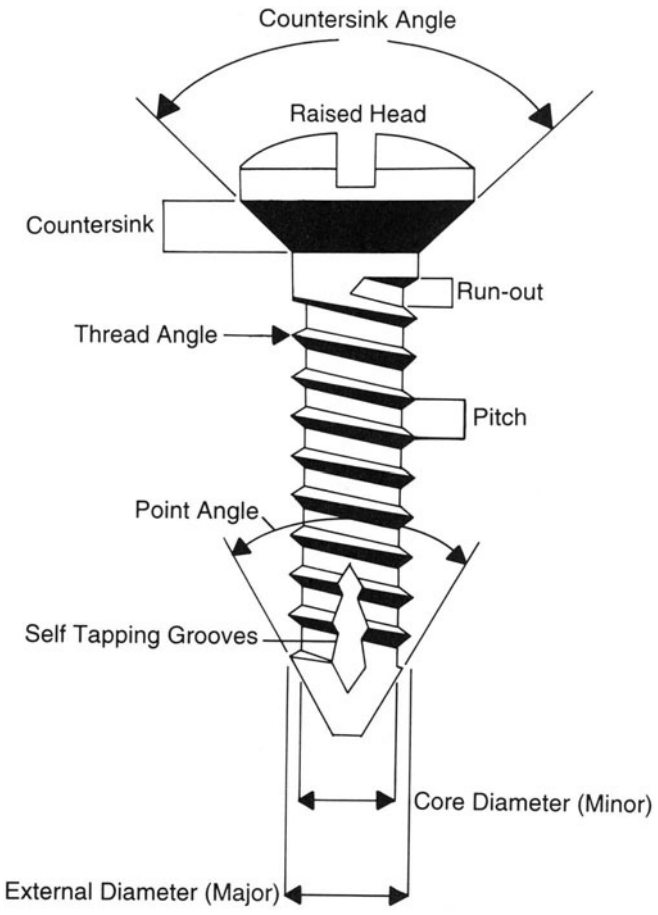

FIGURE 7.17. The anatomy of a screw.

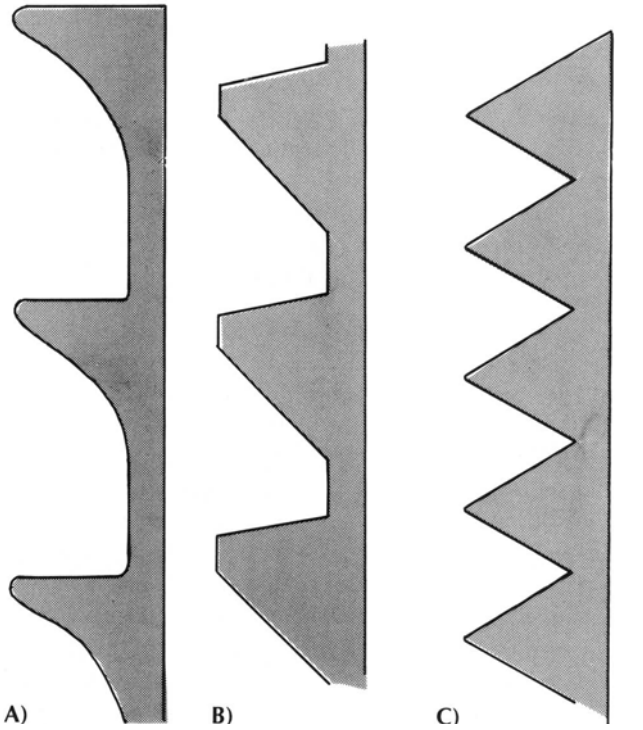

FIGURE 7.18. Screw thread types include modified (A), buttress (B), and v-shaped (C).

more than 90 degrees to the screw shaft.

3. Finally, there are modified screw designs which display slight alterations on the aforementioned configurations.[18]

Screw Application

Screws that require tapping usually have a very blunted round tip and buttress configuration. The pilot hole has to be drilled equal to the core diameter, then by tapping the hole, grooves are cut in the bone identical to the shape of the thread of the screw to be inserted. This type of screw is seldom used nowadays.

Although self-tapping screws were originally v-shaped threaded screws, now self-tapping is applied to buttress type screws as well, because the latter is, indeed, gaining more popularity. Generally, this type of screw has a sharper end and there is a vertical groove within the last three or four threads.

The advantage of the self-tapping screw is that it reduces an additional step of tapping. The disadvantages include reduction in the effective length of the screw as much as 17 to 30 percent, the increased torque required to place the self-tapping screw, more potential for microfractures, a build-up of bone debris during screw insertion, and bone growth into the grooved screw tip, making removal more difficult.[33,34]

Difficulties in the Screw Function

It is very rare for a screw to fail. Generally, it is a failure in proper judgment, which results in screw dysfunction. Some errors include improper choice of drill size; improper placement of the hole by inaccurate or rotational types of drilling, which will result in a wider hole than is needed; and inadequate irrigation, which will result in thermal injury to the bone surrounding the hole with subsequent loosening and failure of the screw. Application of too much torque will result in fracture of the screw, particularly when it is placed in dense bone. Finally, inappropriate screw size for the load is a common error.

It is important to recognize that the holding power of the screw is directly and primarily related to the external diameter (major diameter) of the screw, and is not contingent upon the shape of the screw.

Besides the lagging that was described earlier, the screw can be used as a position screw. With this type of fixation, there is no lagging effect and the screw will be

engaged bicortically (through two segments). The tightening of the screw will only result in individual stabilization of the segments, rather than compressing the bone pieces together. Therefore, it maintains the necessary space in between the segments. This is particularly useful in the stabilization of the sagitally split segment of the mandible during orthognathic surgery, where compression not only is unnecessary, but can also be detrimental and may dislocate the condyle.

Bone Plates

There are two types of plates—the dynamic compression plate and the noncompression plate. The basic principle of the compression plate is the force generated by compression of a spherical plate against an inclined plane. When this spherical surface, which is the screw head, is forced vertically against the inclined surface of the plate, it will move the bone on a horizontal plane. This, in turn, will affect the position of the underlying bone, compressing segments on either side of the fracture against each other when opposing forces are used.

The amount of compression, meaning the horizontal movement, will be dependent on the inclination of the plate and the depth of the plate. This inclination may vary from 16 to 60 degrees. With a smaller degree of inclination, there is more horizontal movement, but less compression force. Conversely, the more the degree of inclination, the less the movement, and the greater the compression force. A 45-degree inclination produces the most efficient force and horizontal movement. It is important to realize that biological compression not only approximates the boney segments, it also enhances bone healing.

Dynamic compression plates are available in sizes 3.5, 2.7, and 2.0 mm. These numbers obviously refer to the external diameter of the screw that is used for fixation. Larger plates are more suitable for mandibular fractures and smaller plates better serve in the fixation of midface fractures.

The eccentric dynamic compression plate is used when a plate is applied in the superior quarter of the mandible. This is to avoid the displacement and separation that is seen when a dynamic plate is applied along the inferior of the mandible. The two nondynamic holes in this four-hole plate lie closest to the fracture. The screws are placed in the nondynamic hole first and tightened to control the bone segments. Next, the screws located in the angled holes of the eccentric dynamic compression plates are tightened, which will approximate the superior border of the mandible.

Noncompression Plate System II

There are two types of plates in noncompression systems, those that are large and those that are small (or miniplates). The large noncompression plates are used for unusual situations, such as when there is a significant bone gap. These plates are also suitable for a comminuted fracture. These are available in 2.7 and 3.5 mm sizes. A variety of plate lengths and shapes are on the market.

Minicompression plates have gained popularity lately. These are available in 1.5 and 2.0 mm sizes and in a variety of styles. The microsystem uses 0.8 mm screws and plates of various shapes and lengths. These plates are available in titanium, stainless steel, and vitallium. Finally, several newer plates with screws and plates slightly larger than the Microsystem have become available.

The Components of Plates and Screws

The three main materials used for surgical screws and plates include titanium, stainless steel, and vitallium. The components of each of these metals are listed in Table 7.1.[18] Each alloy system has specific mechanical properties that govern the performance of that particular plate or screw. These properties are compared in Table 7.2.[18] The terminology used in this table deserves explanation. *Yield strength* refers to the amount of load per unit area required to alter the form of the implant permanently. *Tensile strength* is the load necessary to fracture the material. *Elongation* refers to the changes in the dimension of the metal during mechanical tests, and *elastic modulus* is the ratio of the amount of strength (load per unit area) necessary to produce a change in the shape. This, obviously, means that a rigid material has a higher elastic modulus, whereas a more pliable material will have a low elastic modulus. *Ductility* means the ability to withstand large plastic deformation before failure takes place. The metals with high ductility can be contoured without exhibiting cracks.

Of the available materials, stainless steel has a moderate yield and tensile strength, but is highly ductile. Vitallium is the most commonly used cobalt alloy,

Table 7.1. Element percentages in rigid internal fixation metals.

	Compound			
	Titanium (pure)	Titanium (alloy) (Ti-6Al-40)	Vitallium	316L Stainless steel
Element (percent):				
Aluminum		5.5–6.5		
Carbon	0.1	0.1	0.35	0.03
Cobalt			57.0–68.0	
Chromium			27.0–30.0	17.0–20.0
Iron	0.2–0.5	0.25	0.75	59.0–69.0
Molybdenum			5.0–7.0	2.0–4.0
Nickel			2.5	12.0–14.0
Titanium	98.6–99.6	88.3–90.8		
Vanadium		3.5–4.5		

Table 7.2. Mechanical properties of metals used in rigid internal fixation.

	Titanium (pure)	Titanium (alloy) (Ti-6A1-4V)	Vitallium	316L Stainless steel
Elongation (percent)	25	10	8	40
Tensile strength (1000 PSI)	35	130	95	70
Yield strength (1000 PSI)	25	120	65	40

which is advantageous over stainless steel in many ways. It has a higher yield strength and higher modulus of elasticity, providing more rigidity and strength. This stiffness, however, makes contouring difficult.

Titanium has a low ductility and is weaker than stainless steel and vitallium.

Indications for Bone Fixation

Bone fixation is used when osteosynthesis of fractures, osteotomies, and bone grafts is desired. Proper immobilization of bone fragments is critical for proper healing. If this does not occur, then the possibility of delayed union or even nonunion is quite real. There are many fixation systems that have been used successfully, and there are continually new modifications of these systems that now provide an ample armamentarium to obtain proper healing in various sites and situations.

Craniomaxillofacial Fractures

Although interosseous wiring has been used for many years with relative success in fracture treatment, it has become evident that it provides only two-dimensional stability at the fracture site, and even at its best, does not provide rigid fixation, permitting some flexibility or movement between fragments. With respect to bone healing, this is usually detrimental. As stated earlier, various plate and screw fixation systems, however, do provide three-dimensional stability and true rigid fixation between the fractured bone segments. They come in various sizes depending on the consistency of the bone and the function that it has.

Mandibular Fractures. Treatment of mandibular fractures depends on the status of the teeth, the location and type of the fracture, and the degree of displacement and stability of the bone segments. Traditional teaching states that a favorable nondisplaced fracture in a dentulous patient can be treated either with a soft diet or intermaxillary fixation. More severe or less stable fractures, however, will require internal fixation and in most of these cases, plate and screw fixation will maintain three-dimensional reduction and stability. Displaced fractures of the mandibular angle, associated fractures of the temporomandibular joint that will require early movement and physical therapy, and comminuted fractures will all benefit from mandibular plating. These plates are larger, sturdier plates that will resist the muscle forces of mastication on the lower jaw. Cases in which there is respiratory compromise or where intermaxillary fixation is undesirable will also benefit from plate and screw fixation. In cases of mandibular bone loss due to extensive trauma or tumor ablation, a large malleable reconstruction plate can be used to bridge large defects of the mandible. This can be used alone or in conjunction with bone grafting.

Mid-face Fractures. Fractures of the maxilla, zygoma, and nasoorbital regions can be fixed effectively with smaller plating systems, known as miniplates or even microplates.

These plates are generally noncompression plates that are bent to the proper shape and provide adequate rigid fixation. The advent of lower profile and smaller plating systems, such as the microplating fixation system, has been found to be quite useful in areas around the orbit and the nasoethmoid region. The smaller plates and screws are generally nonpalpable under the skin and the smaller holes and screws are more suitable for better purchase of the fracture segments. These plates have been used widely in infant craniofacial surgery, where the bones between osteotomy sites are very thin and, at times, quite fragile.

Upper Face and Skull Fractures. Fractures in the upper face and skull can be treated, generally, with mini- or microfixation systems. Sheets of mesh are now available that can bridge large fracture defects or large bone graft donor site defects. These sheets of mesh have also been used to reconstruct the orbital floor, with or without bone grafting.

Orthognathic Surgery. Orthognathic surgery involves osteotomies in the upper and lower jaw bones to correct abnormalities of facial proportions. In the mandible, segmental sagittal splits and other osteotomies can be fixed with lag screws or plates and screws maintain proper position and alignment. Similarly in the upper jaw and midface, Laforte I, II, or III osteotomies are fixed with plates and screws that have been able to reduce the incidence of relapse of these bone segments over time.

Bone Grafts. An increasing amount of literature now available shows that proper fixation of bone grafts is necessary to improve the longevity and decrease the amount of resorption of these grafts. Lag screws, plates and screws, or washers and screws can all be used to properly fix bone grafts.

Lag Screw Technique

Lag screws are used in cases of oblique fractures, osteotomies and fixation of bone grafts, and can provide excellent fixation and compression. Caution should be used

in the case of sagittal split osteotomies, because the condyle may be displaced when the lag screws are tightened securely and the inferior alveolar nerve can be pinched or even crushed in between the lateral and medial segments. The outer cortex of bone is overdrilled with a larger drill bit so that the threads of the screw do not engage, then the distal bone cortex is drilled with a smaller drill bit so that the screw threads do engage and, ultimately, compress the bone segments together. Bone grafts are very well suited to this technique; however, at least two lag screws are necessary to avoid rotation of the graft.

Compression Plating

The concept of compression is fully described in the osteosynthesis of long bones and is useful in providing primary bone healing in these situations. Because most of the craniomaxillofacial skeleton undergoes a form of primary bone healing after fracture, this is usually not a necessary component to fracture fixation at these sites. It can aid in certain situations to bring bone segments closer together and in better alignment. This may be useful in mandibular fractures, where distraction forces may be quite strong. Eccentric dynamic compression was designed to provide adequate rotation of mandibular fracture segments that give, in turn, better alignment of the alveolar segments of the bone. This can also be accomplished by placing the patient in intermaxillary fixation or with the application of a tension band on the teeth at both sides of the fracture site.

Fractures in Children

Pediatric facial trauma has certain characteristics that distinguish it from the adult. Differences include the smaller face to skull ratio, the paucity of aerated sinuses, the presence of mixed dentition, and the overall flexibility of bones in comparison to an adult. Although adequate reduction of fractured segments is as desirable in children as it is in adults, many times greenstick fractures are present that may not require rigid fixation for proper healing. Furthermore, rigid fixation may have a deleterious effect on the growth of the very actively changing structures of the pediatric face and skull. Studies still need to be conducted to fully define the ill effects or disturbances in growth due to fracture fixation. Many times simple wire fixation or microplating may be all that is necessary in these pediatric fractures. Care should always be taken not to injure toothbuds when applying fixation devices. The ability of the young craniomaxillofacial skeleton to remodel is great and can be helpful in the long-term management of these injuries.

Advantages and Disadvantages of Bone Fixation Systems

The disadvantages include the more permanent placement of a foreign material, even though it has not been proven to be consequential, particularly with materials such as vitallium and titanium. Another disadvantage of plates and screws is the additional expense. However, when one considers the savings related to a reduction in hospital stay, the shorter recovery period, and earlier gainful function, the economic difference may not be significant and rigid fixation might even be advantageous. Another disadvantage of rigid fixation, especially when it is compared to other techniques of fixation, is the necessity of power equipment. Should there be a misjudgment in the selection of the proper screw and plate profile, these fixation devices can become palpable or visible.

The advantages of rigid fixation, regardless of the type of plate used, include the ability to stabilize the bone fragments. Even with the miniplates, a significant degree of stabilization can be achieved that will not be overcome by the masticatory muscles. However, the rigidity that is achieved through larger plates is not completely matched by the smaller plates. Both large and small plates can be placed transorally, but the smaller plates are more suitable for this approach. With the use of plates, it is possible to avoid or at least minimize the period of postoperative intermaxillary fixation. There is more airway safety following both orthognathic surgery and fixation of fractured segments. Often a tracheostomy can be avoided because of the security that rigid fixation provides.

The additional benefit of rigid fixation on healing cannot be underestimated. This becomes even more critical when there are comminuted fractures, which ordinarily are prone to complications such as nonunion, malunion, or infection. In the case of orthognathic surgery, an additional advantage, besides the ability to resume normal eating habits sooner, is earlier resumption of the orthodontic work, leading to faster completion of objectives. Another decided advantage of rigid fixation over other types of fixation of fractures is a reduction in hospital stay.

Absorbable Rigid Fixation and Screws

Recent information concerning the undesirable characteristics of rigid fixation devices (such as palpability, late infection and, more importantly, limitation of growth in infants), has evoked a great deal of interest in the development of absorbable rigid fixation systems, screws, and plates. Although in a preliminary state, the results from clinical and research investigations appear to be very promising. The long-term results, however, will not be available for many years.

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CHAPTER 8

General Evaluation of the Facial Trauma Patient

William A. Crawley

Fifty to seventy percent of patients with facial injuries will have an injury to other organ systems.[1–3] Patients with maxillofacial injuries may occasionally be rapidly assigned to the plastic surgery service, and it is, therefore, incumbent upon the plastic surgeon to ensure proper multisystem evaluation. A systems evaluation should be undertaken, such as the following, which is used at the Maryland Institute of Emergency Medical Services Systems[4]:

1. Airway, respiration, fluid resuscitation, and control of hemorrhage.
2. C-spine injury (films must include C1–C2 and C6–C7. If not able to be visualized, the patient must be assumed to have a C-spine fracture and should have a cervical collar, and movement of the head should be restricted).
3. Upright chest x-ray (pneumothorax, hemothorax, ruptured thoracic aorta, rib fractures, pulmonary contusion, and aspiration).
4. Head injury
   A. Glasgow coma scale is evaluated and consists of:
      1. Level of consciousness.
      2. Eye-opening response.
      3. Ability to speak (briskly purposeful, spastic, flaccid, etc.), can't move, can't talk, can't open eyes.
   B. CT scan.
   C. Intracranial pressure monitor, if comatose, or coma scale less than 15 if general anesthesia is to be used.
5. Abdominal injury (peritoneal lavage).
6. Extremity injury, pelvic injury (appropriate x-rays should be routine to detect occult fractures).

Emergency Treatment of Maxillofacial Injuries

Emergency treatment of maxillofacial injuries consists of management of life-threatening facial emergencies. These are airway obstruction, bleeding, and aspiration.

Airway Obstruction

Injuries that compromise the nasal, oral, or tracheal airway may cause obstruction and result in death. Fractures of the nasoorbital complex, fractures of the maxilla, and fractures of the mandible may all lead to a compromised airway. Laryngeal or tracheal injuries and significant facial burns may also result in swelling with the inability to breathe. Foreign objects such as avulsed teeth, dentures, and bridgework may also cause airway obstruction. Penetrating trauma to the oral facial region, especially the floor of the mouth or neck, may result in significant swelling and airway obstruction. Respiratory obstruction may occur rapidly; therefore one should anticipate airway obstruction before it becomes an acute emergency and provide an adequate airway by endotracheal intubation or tracheotomy. Cricothyroidotomy is the preferred surgical treatment in the event of an emergency airway obstruction.

In patients with maxillofacial injuries who will require operative treatment, the indications for tracheotomy versus endotracheal intubation relate to the ease of management of facial fractures. Tracheotomy or endotracheal intubation is usually performed in the following patients: (1) pan facial fractures (combined nasal, maxillary, and mandibular fractures); (2) the multiple fractured mandible (when the tongue falls back occluding

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the pharynx with swelling of the floor of mouth and neck); (3) massive soft tissue swelling; (4) the use of a headframe with rigid fixation of the mandible to the cranial vault; (5) laryngeal or tracheal injury; (6) intermaxillary fixation in patients with significant head or chest injuries; and (7) the possibility of a difficult reintubation.

When sufficient time exists, an elective tracheotomy may be undertaken. The patient is placed in a dorsal recumbent position with the head and shoulders being placed on a roll to extend the neck. It is essential that the patient be cleared of a cervical spine injury prior to this maneuver. A transverse incision is made two fingersbreadth above the suprasternal notch and should fall within a skin crease. The incision is taken down to the fascia between the strap muscles, where a vertical fascial incision separates the strap muscles in the midline. Absolute hemostasis must be ensured, including dividing the thyroid isthmus if indicated. The lower portion of the larynx and cricoid cartilage should be identified. A vertical incision is planned through the third and fourth tracheal rings. Prior to making the incision, a large nonabsorbable suture is passed on either side of the trachea so that when the incision is made, one has complete control of the opening. A tracheostomy tube is then inserted into the trachea under direct vision. The wound is then gently closed, the flanges of the tracheostomy tube sutured to the skin, and a loose tie placed around the neck.

The postoperative complications of tracheotomy may include secondary hemorrhage (including innominate artery erosion), pneumothorax, tracheal stenosis, dislodgement of the tracheostomy tube, subcutaneous emphysema of the neck, and tracheoesophageal fistula.

As previously mentioned, a cricothyroidotomy (coniotomy) may be performed in case of an extreme emergency. This procedure is technically easier and faster to perform; however, it is only a temporizing measure. A horizontal incision is made between the thyroid and cricoid cartilage and the cricothyroid membrane divided. A tube is then placed into the trachea. An elective tracheotomy may be carried out at a later time.

Hemorrhage

Life-threatening hemorrhage may result from facial lacerations or from closed maxillofacial injuries. Bleeding from facial lacerations should initially be controlled with digital pressure until the exact location of the bleeding vessel is identified. Great care must be taken not to injure any of the branches of the facial nerve. Profuse bleeding may also occur with any fracture communicating with the nose. These include fractures of the nasal bones, sinuses, LeFort fractures, cranial base fractures and fractures of the nasoethmoid, zygoma, and orbital floor or medial orbital wall connecting with the sinuses. Bleeding from closed maxillofacial injuries are usually due to lacerations of arteries and veins in sinus cavities.

Four techniques are recognized for the control of profuse hemorrhage:

1. *Anterior and posterior nasal packing* (Fig. 8.1). This is the most efficient means of controlling profuse nasal bleeding. A posterior nasal pack may be placed, or two Foley catheters may be placed, through the nose into the posterior pharynx, inflated, and secured against the posterior choanae. The anterior nose is then packed with vaseline-impregnated gauze. If bleeding continues and the posterior pack is firmly in place, the anterior pack should be removed and repacked. One must avoid excessive pressure on the columella for fear of necrosis. A period of 2 hours is usually sufficient to control bleeding, after which time the pressure on the columella can be relaxed. The packing is usually removed within 24–72 hours, especially if a CSF leak is noted.
2. *External compression dressing*. A Barton and circumferential bandage may be used as an external compression dressing. This manuever is usually temporary and rarely necessary.
3. *Reduction of facial fractures*. Reduction of the involved facial bone fracture may help control bleeding.
4. *Arterial ligation*. When hemorrhage has not been controlled by any of the above measures, arterial ligation may be necessary. This may include bilateral external carotid and superficial temporal artery ligation. More selective arterial ligations such as the internal maxillary or ethmoid artery may be performed; however, these procedures are technically difficult and may not be possible in the face of profuse hemorrhage.

It should be remembered that in patients with profuse hemorrhage, appropriate blood replacement by a transfusion as well as assessment of depleted coagulation factors are necessary. Coagulopathies are common in patients with massive bleeding.

Aspiration

Pulmonary aspiration of blood, saliva, and gastric contents may be seen with maxillofacial trauma, especially if a head injury is present. Significant bleeding may be seen with LeFort and mandibular fractures, and placement of a cuffed endotracheal tube or tracheostomy tube may prevent aspiration in these patients. Clinically, patients who have aspirated may have noisy respirations in addition to low arterial oxygen content and decreased lung compliance. A chest radiograph may show an infiltrate. Tracheal suction should be performed and the aspirated material identified. Treatment will depend upon the aspirate.

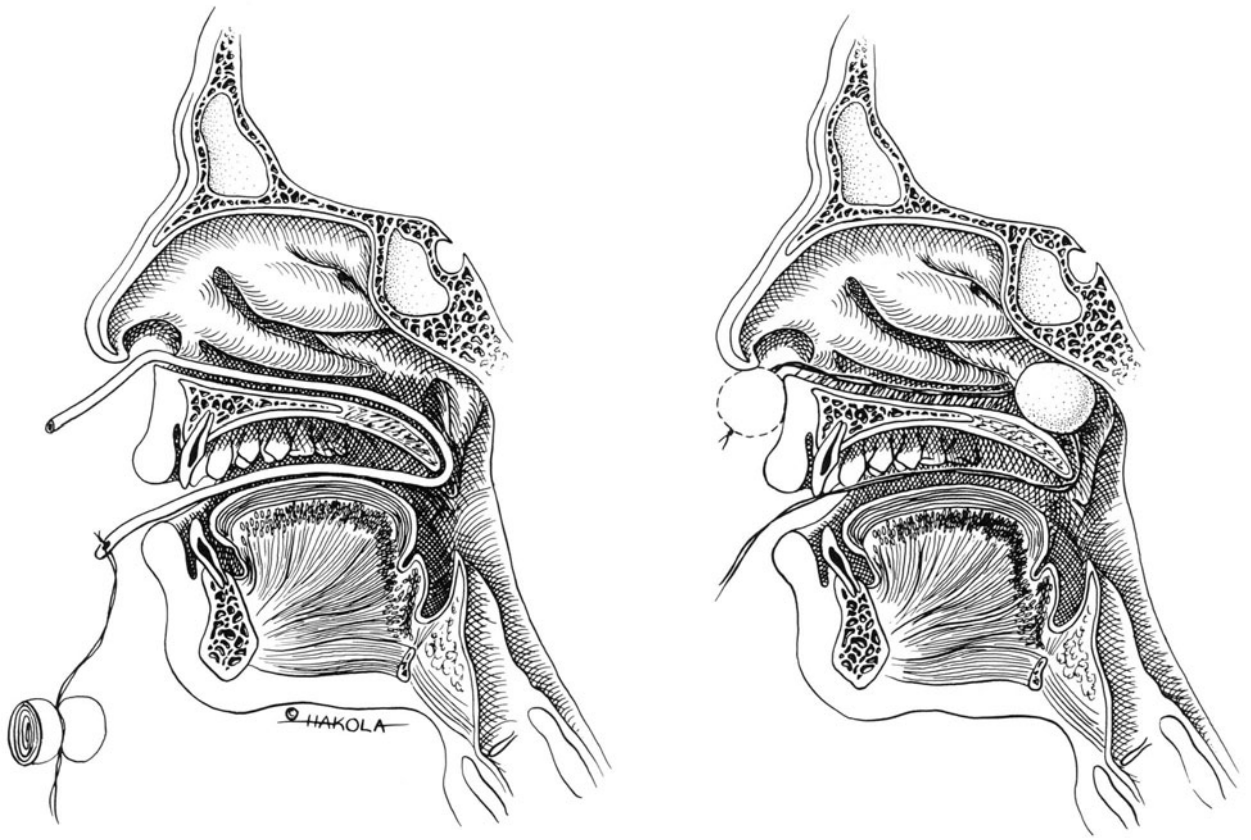

Figure 8.1. Anterior and posterior nasal packing. A catheter is placed through the nose into the posterior pharynx and back out through the mouth. The posterior nasal packing is attached and the catheter pulled up against the posterior choanae. A Foley catheter may be used in place of the posterior pack. The anterior nose is then packed with Vaseline impregnated gauze.

Accompanying Injuries

Cervical spine and intracranial injuries frequently accompany maxillofacial trauma.[5–7] The most frequently missed cervical spine injuries are those located high or low.[7] A CT scan should be performed to rule out these injuries. Management of facial fractures may be limited in terms of exposure because of a cervical spine injury. Intracranial injury may be seen, especially with injuries of the upper facial skeleton.[8–11] A CT scan should be performed in all of these patients. If a general anesthetic is needed in a patient who has a significant intracranial injury, an intracranial pressure-monitoring device should be used. Acute facial fracture management is usually limited in patients with pressures greater than 25 torr.

Clinical Examination

Once emergent conditions have been managed, attention may be turned to the definitive diagnosis and treatment of the maxillofacial injury. A thorough clinical examination will frequently provide the diagnosis, with radiographic evaluation confirming the clinical diagnosis. Any patient should be suspected of a facial injury if any one of the following is noted: contusions, bruises or discoloration, lacerations, pain, paresthesia, visual disturbance, or facial deformity.

The clinical examination of the face should progress in an orderly fashion. One should check for symmetry and deformity as well as palpate all bony surfaces. Evaluation of facial nerve function as well as sensation should be noted. Intraoral examination is absolutely essential as is the wearing of gloves during this portion of the examination. One should look for intraoral lacerations and bruises as well as for malocclusion. Fractures of the dentoalveolar complex, as well as fractured, loose, or missing teeth should be noted. Mandibular movement should be checked for opening and possible deviation of the mandible. Pain may be present with opening or excursive movements. The head of the mandibular condyle may be palpated with the examiner's finger inserted within the external auditory canal and asking the patient to open. Midface fractures may have mobility as well as malocclusion (Fig. 8.2). Nasal examination should include checking externally for nasal bone displacement as well as intranasally looking for a septal hematoma. Examination of the eyes and orbits should include evaluating the eyelids for injury, the possibility of corneal penetration, pupillary size and symmetry, visual acuity, and the presence of double vision or loss of vision. The combination of periorbital hematoma and subconjunctival hemorrhage suggests an orbital fracture (Fig. 8.3). Extraocular movements should be

Figure 8.2. Mobility of the midface is checked with the cranium stabilized by attempting to move the anterior maxilla. Reprinted with permission from McCarthy. *Plastic Surgery*. Philadephia: WB Saunders; 1989, Vol. 2: p. 883.

Figure 8.3. Periorbital ecchymosis and subconjunctival hemorrhage suggest an orbital fracture.

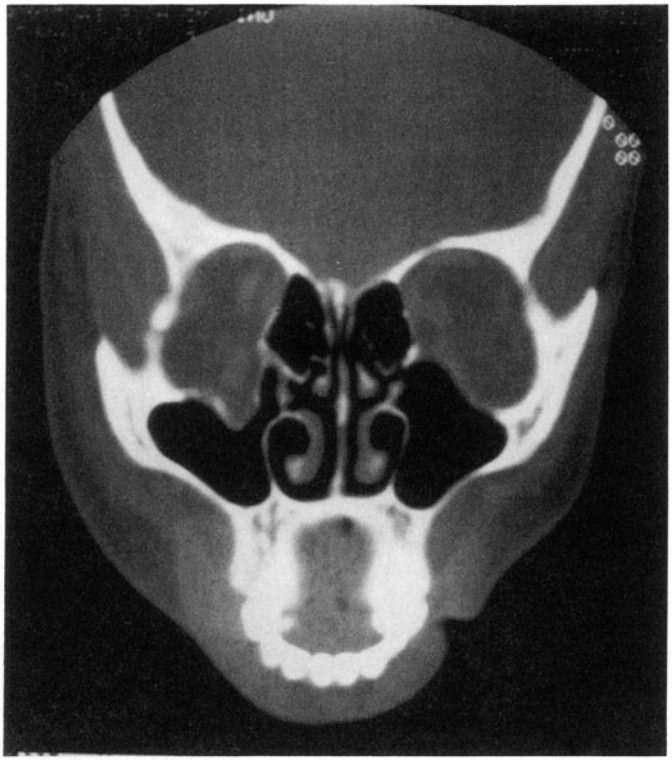

Figure 8.4. Coronal CT scan demonstrating fracture of the orbital floor with herniation of orbital contents into the maxillary antrum.

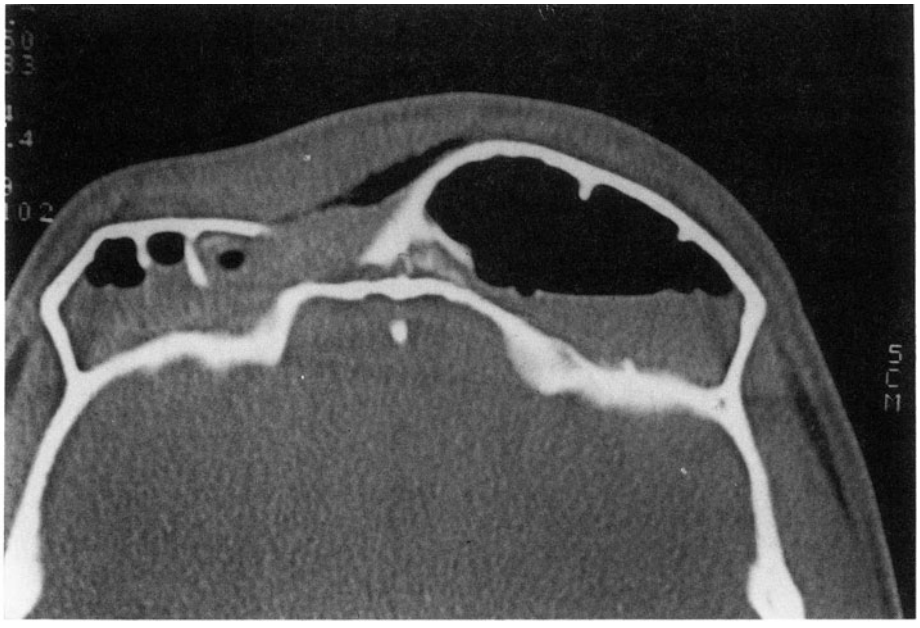

Figure 8.5. Axial CT scan showing displaced anterior wall fracture of a large frontal sinus.

checked in all fields of gaze; appropriate ophthalmologic consultation should be obtained. CSF leakage may be seen with fractures of the cribriform plate, and bleeding from the ears suggests a basilar skull fracture.

Radiographic Examination

Radiographic evaluation of the maxillofacial injured patient may consist of plain radiographs, CT scans, and specialized dental radiographs, such as the Panorex and periapical films.

Although plain radiographs have been to a great extent replaced by CT scans, the best views include the Water's view, the Towne's view, the posteroanterior (Caldwell) view, and lateral oblique films of the mandible.

The CT scan has become extremely valuable in the radiographic evaluation of facial bone injuries, especially those of the upper facial skeleton (Figs. 8.4 and 8.5). Any patient suspected of a fracture of the frontal sinus, supraorbital rim, orbit, nasoethmoid region, and LeFort area should have a CT scan performed with thin cuts.

A Panorex radiograph is most helpful in identifying fractures of the ramus of the mandible and the body of the mandible. The symphysis region is sometimes obscure on these films. Periapical radiographs are helpful any time a tooth is noted in the line of fracture.

Dental Impressions and Models

Dental models provide an unequaled means of studying the patient's dental occlusion. Alginate impressions should be obtained on any patient with a jaw fracture. Dental stone is used to construct the models. A wax bite impression is used to accurately determine the patient's intermaxillary relationship.

One should check the dental models for rotated and fractured teeth, arch alignment, dental midline, and first molar relationship. Checking for wear facets may be very helpful in a patient with a preexisting malocclusion.

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CHAPTER 9

Fractures of the Mandible

William A. Crawley and Allan J. Sandel

The mandible is one of the most frequently fractured facial bones along with the nose and zygoma, and the most frequently fractured bone in the multiple-injured patient. Mandibular fractures account for approximately 20 percent of all facial fractures. Motor vehicle accidents and physical altercations are the most common causes of fractures of the mandible. Areas of weakness in the mandible are the neck, the subcondylar region, and the angle; these will be the most frequently seen areas to fracture. The presence or absence of teeth will also contribute to the location of the fracture. Greater than one half of all mandible fractures are multiple; therefore, one should always look for a second site of fracture. Mandibular fractures may be open or closed either through the skin or intraorally through mucosa (compound fractures).

Muscle Groups Associated with the Mandible

In treating fractures of the mandible, it is important that one be knowledgeable with regard to the origin, insertion, and action of muscles attached to the mandible (Fig. 9.1). Displacement of mandibular fractures depends upon the direction of muscle pull; therefore, muscle groups that attach to the mandible may be divided according to the displacement produced.

Suprahyoid Muscles and Mandibular Depressors

The anterior or suprahyoid muscle group (Fig. 9.1A and 9.1B) consists of the geniohyoid, mylohyoid, and digastric muscles. These muscles act to rotate the mandible downward to initiate opening the mouth. The geniohyoid muscles arise from the inferior mental spine and inserts into the body of the hyoid bone. The mylohyoid muscle is attached to the inner surface of the mandible along the internal oblique or mylohyoid line and extends to the hyoid bone to form the muscular floor of the mouth. The anterior belly of the digastric muscle extends between the inner side of the lower border of the symphysis and a ligamentous attachment to the lateral cornu of the hyoid bone. The posterior belly of the digastric muscle extends between the mastoid notch of the temporal bone and the hyoid bone. The genioglossus muscle originates from the superior mental spine on the inner surface of the body of the mandible, spreads out in a fan-like fashion, and is inserted into the body of the tongue and the upper surface of the hyoid bone.

Muscles of Mastication

The posterior group of mandibular muscles consists of the muscles of mastication (Fig. 9.1C). The muscles of mastication are the masseter, temporalis, medial pterygoid, and lateral pterygoid.

The masseter muscle arises from the lower border and deep surface of the whole zygomatic arch. The anterior two thirds of the muscle runs down and back to insert into the lateral ramus of the mandible close to its lower border. The posterior third of the muscle runs down with a slight anterior inclination to insert deep to the anterior part into the upper part of the ramus; a few of its fibers insert into the capsule of the temporomandibular joint. The masseter muscles elevate and pull the mandible upward and forward.

The temporalis muscle arises from the skull over the temporal fossa as well as from the temporal fascia, which is superficial to it. From this origin, the muscles narrow to a flat tendon beneath the zygomatic arch,

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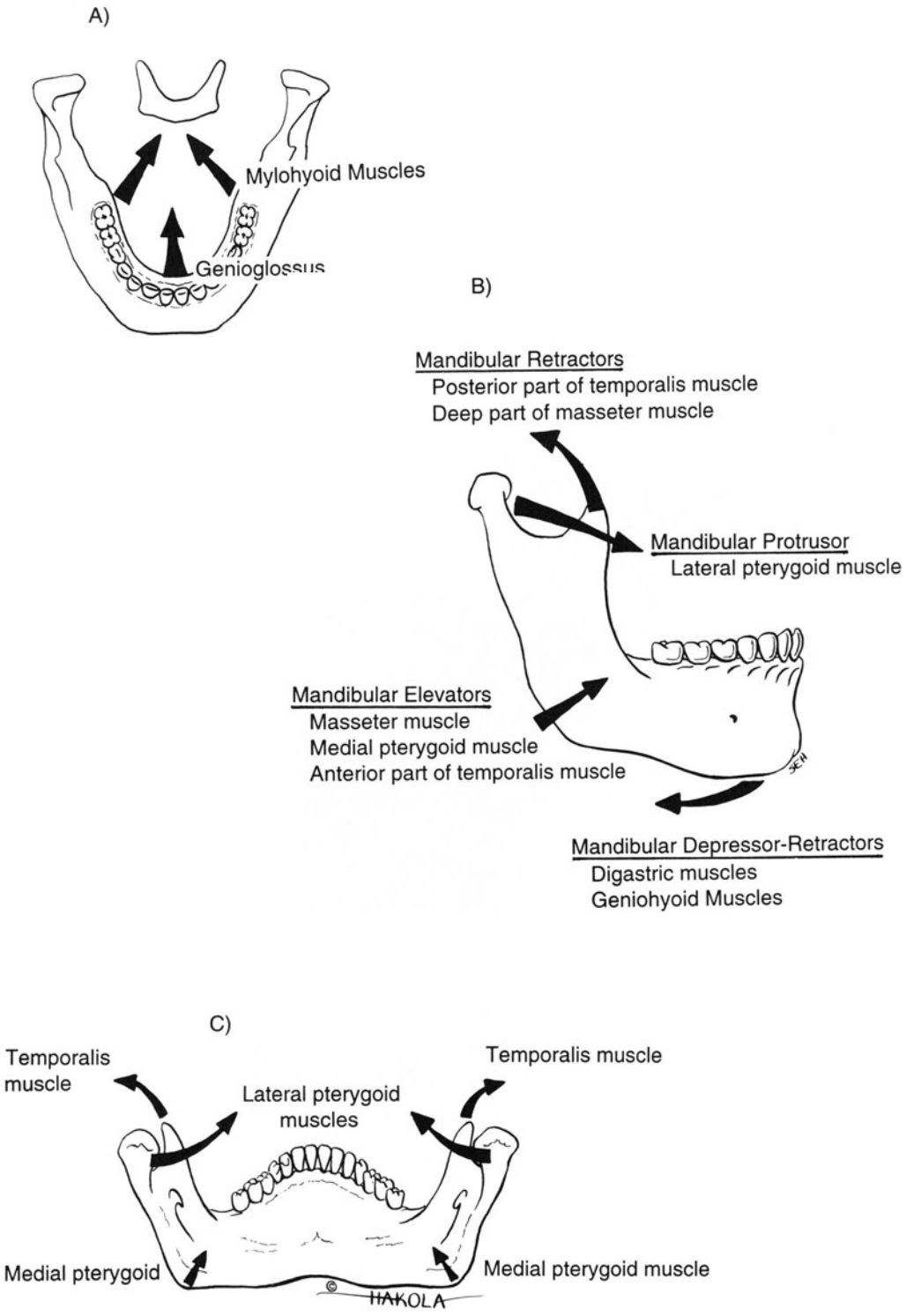

Figure 9.1. Muscle groups attached to the mandible. (A) Anterior muscle group. (B) Posterior muscle group and mandibular depressor-retractors. (C) Posterior muscle group.

which inserts into the margin and deep surface of the coronoid process of the mandible and along the anterior surface of the ramus in its upper portion. The anterior fibers are elevators and the posterior fibers are retractors of the mandible.

The medial pterygoid muscle arises from the medial surface of the lateral pterygoid plate. It runs downward, backward, and laterally to insert into the deep aspect of the lower third of the ramus of the mandible and forms a sling along with the masseter muscle (pterygomasseter sling). The function of the medial pterygoid muscle is to assert upward, medial, and forward traction on the mandible.

The lateral pterygoid muscle has a broad origin by two heads, the pterygoid head from the lateral surface of the lateral pterygoid plate and the sphenoidal head from the adjacent part of the undersurface of the skull. The two heads pass backward and laterally, tapering to insert by a strong tendon into the front of the neck of the mandible as well as into the capsule of the temporomandibular joint. The upper portion pulls the mandible upward, medial, and forward; the external portion pulls the condyle downward, medial, and forward. Contraction of the lateral pterygoid on one side pulls the mandible to the opposite side. Contraction of both lateral ptergoid muscles simultaneously protrudes the mandible.

Mandibular Bone

It is important to understand that the mandible is a bilateral bone connected at its midline (symphysis). It is unable to move independently of its opposite side. The mandible articulates with itself with respect to its bilateral temporomandibular joints and also with the maxilla by the dental occlusion between the maxillary and mandibular teeth.

Anatomic reduction in every dimension is extremely important in order to reestablish the preinjury skeletal relationship of the mandible to itself and with the maxilla.

Diagnosis and Treatment

The diagnosis and treatment of mandibular fractures will be discussed by site, including the condyle, neck, ramus, angle, body, parasymphysis, and symphysis regions. It should be emphasized that the most important goal in treating fractures of the mandible is to reestablish the patient's preinjury dental occlusion. If the preinjury dental occlusion is reestablished, then the skeletal relationship between the components of the fracture will readily be placed into anatomic reduction.

Condylar Fractures

Patients with condylar fractures (Fig. 9.2) may present with pain or limitation of movement, tenderness in the preauricular region, malocclusion or deviation of mandibular opening, or blood in the external auditory canal. Frequently, a history is given of an impact upon the symphyseal region of the mandible. In addition to these signs and symptoms, one should palpate the head of the condyle, with the examiner's finger being inserted into the external auditory canal and asking the patient to open, or having the examiner manually distract the body of the mandible in the obtunded patient. One should feel the condylar head of the mandible moving equally on both sides. Radiographs most helpful in diagnosing fractures of the mandibular condyle include the Panorex, the Towne's view, and especially the CT scan.

The typical clinical finding of a unilateral displaced condylar fracture is a dental malocclusion, in which the patient is occluding prematurely on the maxillary and mandibular posterior teeth on the side of the fracture (Fig. 9.2). The reason for this premature occlusion is due to a loss of the vertical height of the mandibular ramus on that side. In patients who suffer from a bilateral displaced condylar fracture, there is bilateral premature occlusion between the maxillary and mandibular posterior teeth, thus leading to an open bite deformity, in which the patient is unable to bring the incisal edges of the maxillary and mandibular anterior teeth together. In the bilateral displaced condylar fractures, there is a bilateral decrease in the vertical height of the rami of the mandible (Fig. 9.2).

Treatment of fractures of the mandibular condyle may be divided according to the line of attachment of the capsule, thereby producing either an intracapsular (condylar head) fracture or an extracapsular (condylar neck or subcondylar) fracture (Fig. 9.3). Treatment of mandibular condylar fractures are controversial. Generally one should be conservative, with the goal being joint motion, reestablishing normal occlusion, and preserving the vertical height of the ramus. Intracapsular fractures are generally considered best managed conservatively, as these fractures are usually comminuted, and it is frequently difficult to technically do anything with these fractures. If conservative (nonoperative) treatment produces an unsatisfactory result, condylar replacement may be contemplated at a later date. Extracapsular fractures of the condyle involve the neck and subcondylar region. These fractures are generally treated closed (with certain exceptions as noted below). One would generally place the patient in intermaxillary fixation for 2 weeks followed by opening with close observation of the occlusion, and perhaps using guiding elastics and elastic intermaxillary fixation at nighttime. Physical therapy is essential.

In the course of treating fractures of the condylar area, there may be a time when an intraarticular or subcondylar fracture is noted by history and radiologic examination; however, per clinical examination, the patient will

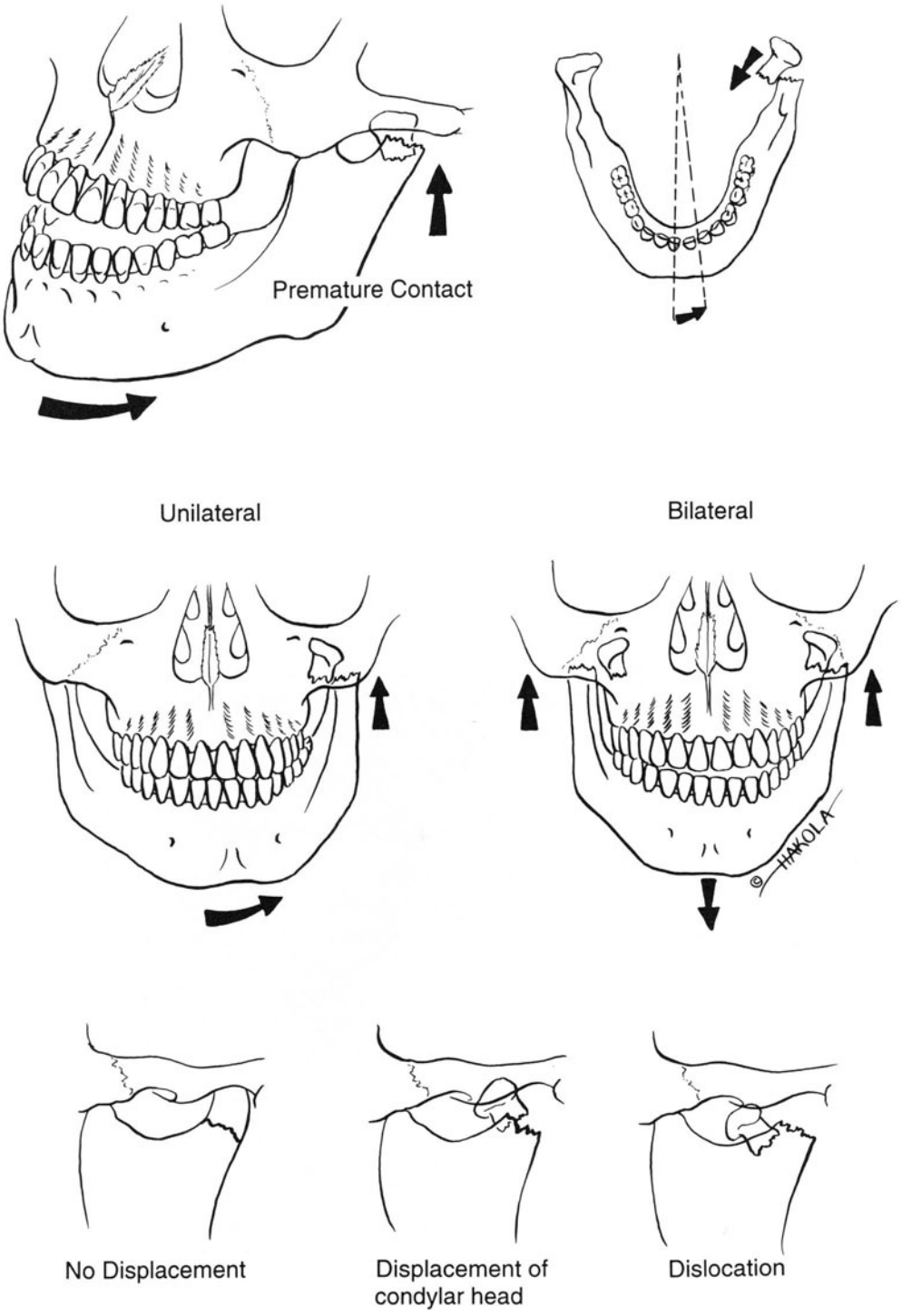

FIGURE 9.2. Condylar fractures.

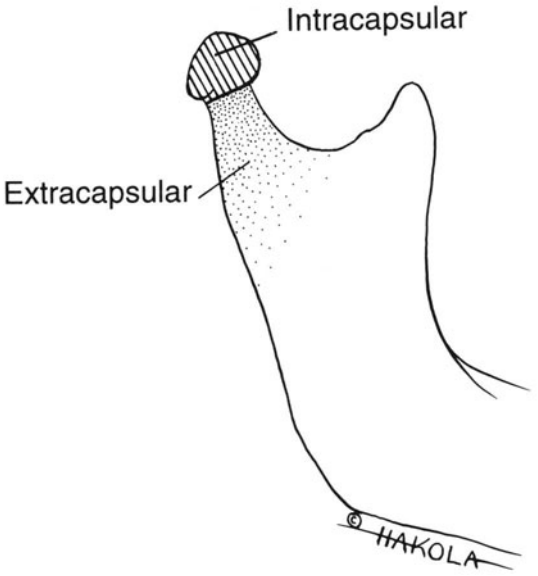

FIGURE 9.3. Anatomic division of condylar fractures.

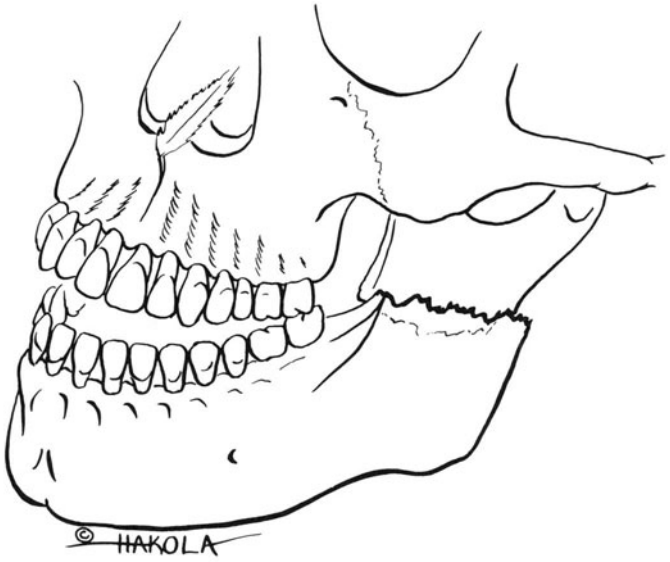

FIGURE 9.4. Ramus fracture.

have a normal dental occlusion. When this is encountered, there is a tendency toward treating the apparent undisplaced condylar fracture with analgesics and liquid diet without closed reduction and intermaxillary fixation. The physician should be aware that normal mandibular movement with regard to swallowing and speech may cause a displacement by pulling of the lateral pterygoid muscle and eventual displacement of the proximal segment. Thus, what may have been diagnosed as an undisplaced fracture of the condylar area, if not treated appropriately, may result in a displaced condylar fracture. The conservative approach in this type of injury would be to place the patient in intermaxillary fixation with closed reduction.

Open reduction of fractures of the mandibular condyle and neck are technically difficult as the anatomy in this area is quite complex. Some general indications for considering open reduction include: (1) fragments causing functional interference with opening; (2) bilateral fractures with persistent open bite from fracture malposition; (3) limitation of movement of the jaw from the condylar position; (4) fracture, dislocation of the condylar head, medially or laterally out of the glenoid fossa or superiorly into the medial cranial fossa; and (5) bilateral subcondylar and LeFort fractures. A combined preauricular and modified Risdon approach generally provide the best exposure for open reduction. When open reduction of a mandibular condylar fracture is performed, some type of rigid internal fixation is always necessary. A small plate or lag screw provides the best stabilization. Joint mobility and proper occlusion once again is the goal. Generally, a 2-week period of intermaxillary fixation would be indicated prior to beginning movement of the mandible, with subsequent aggressive physical therapy and close observation.

Ramus Fractures

Fractures of the ramus (Fig. 9.4) may include the coronoid process as well as sagittal and horizontal fractures of the ramus. These fractures are much less common. The diagnosis is made by tenderness in the area, limited opening, and possible malocclusion. Radiographically, the Panorex, external oblique views of the mandible, and the CT scan are helpful. Fractures of the coronoid process may not produce a malocclusion, and conservative treatment may be all that is necessary. The goal in treatment of ramus fractures is reestablishing the patient's occlusion and facial height. Minimally displaced fractures may be treated with intermaxillary fixation. Fractures that are more displaced and produce malocclusion or reduced facial height may require internal plate and screw fixation, which is usually via intraoral approach.

Angle Fractures

The angle of the mandible is a frequently fractured area, as this is a thinner portion of bone and frequently the third molar tooth may be in this region. The pterygomasseteric sling and temporalis muscle tend to pull the proximal segment upward, whereas the suprahyoid musculature tends to displace the distal segment inferiorly and medially (Fig. 9.5). Clinically, one will note ten-

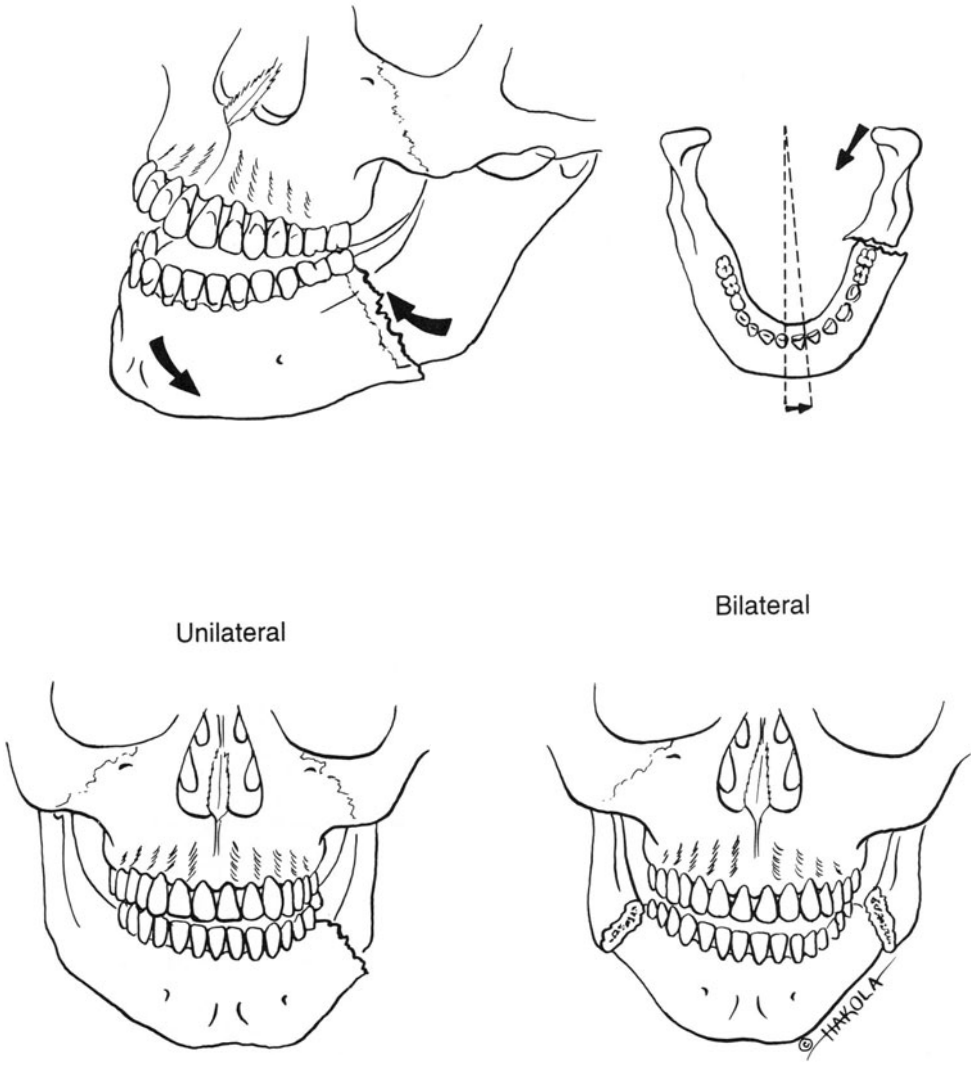

Figure 9.5. Angle fractures.

derness over the fractured area as well as pain with opening and possible malocclusion. There may also be mobility of the fracture segments. Displacement is from the pterygomasseteric sling, which tends to pull the proximal segment upward. Attention should be paid to the presence or absence of the mandibular third molar as well as the degree of soft tissue and bony impaction. If any doubt exists as to whether the tooth should be maintained, a general rule is to remove the third molar tooth. Teeth in the line of fracture will be discussed later in the text.

Nondisplaced or minimally displaced angle fractures may be treated with intermaxillary fixation with close observation for possible subsequent displacement. Fractures of the angle distal to the last tooth in the dental arch should be maintained by interosseous fixation, as the application of arch bars for closed reduction will not encompass both sides of the fracture. Therefore, the posterior segment of bone will not be stable. An open reduction may be performed either through a modified Risdon or submandibular incision or from an intraoral approach. Once the fracture is reduced, fixation may be either with a lag screw technique or the use of a plate and screw system. When a mandibular plate is used along the inferior border, a second plate is usually placed at the superior border to act as a tension band (Fig. 9.6). Generally, the larger, inferior border plate should have three screws in each fragment on either side of the fracture site.

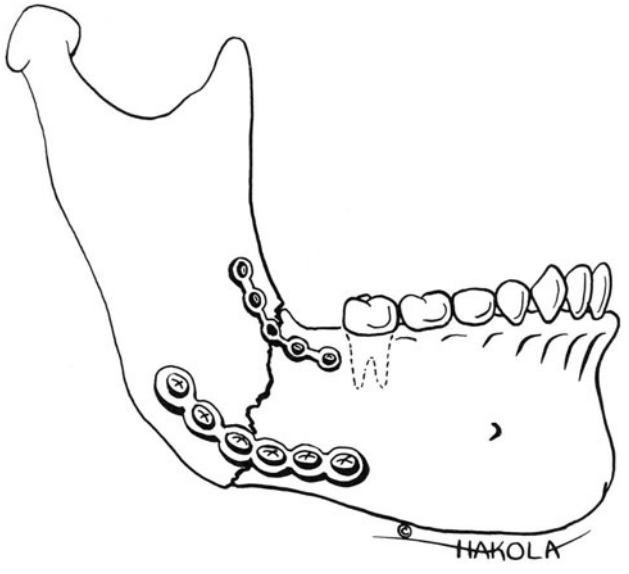

FIGURE 9.6. Superior border plate acting as a "tension band."

Body Fractures

Fractures of the mandibular body may be diagnosed by tenderness of the region, pain with opening, malocclusion, and mobility. The pterygomasseteric sling, temporalis, and lateral pterygoid muscle will displace the proximal segment upward and medial. The mylohyoid and geniohyoid muscles will deflect the distal segment downward and medial (Fig. 9.7). Additionally, one may note an intraoral communication with a laceration of the gingiva or mucosa. Radiographic evaluation may consist of a Panorex, plain film, or CT scan. It is imperative that any tooth in the line of fracture be thoroughly evaluated radiographically. (Further consideration of teeth in the line of fracture is discussed later.) In general, nondisplaced or minimally displaced fractures of the mandibular body may be managed with intermaxillary fixation and close observation. More displaced fractures may require internal fixation. An intraoral or submandibular approach may be used with a plate and screw system along the inferior border of the mandible, once again using three screws in each segment on either side of the fracture. The superior tension band is provided by the arch bar.

Symphysis and Parasymphysis Fractures

The symphysis and parasymphysis regions are quite strong and less frequently fractured, as the symphyseal area has a great deal of cortical bone. Fractures of the symphysis require a great deal of force; therefore, one should always examine for condylar fractures and hyperextension cervical spine injuries. The diagnosis is made by tenderness over the area, pain with opening, malocclusion and mobility (Fig. 9.8A). Radiographic evaluation consists of plain films, CT scans, and a submental vertex or occlusal view. The Panorex radiograph is not always reliable for the symphysis and parasymphysis regions. Once again, intermaxillary fixation may be used to treat nondisplaced or minimally displaced fractures; however, close observation is needed because of significant muscle pull on the anterior mandible. A large inferior border plate or a lag screw (Fig. 9.8B and 9.8C) may be used to treat fractures in this region. Oblique fractures may create overriding of the bony fragments; therefore, one should be most mindful of establishing the patient's preinjury occlusion. Generally an intraoral approach is adequate. The mental nerve should be identified and protected, as well as making sure that the tooth roots are not injured by the operator.

Multiple Mandibular Fractures

As previously noted, over one half of mandible fractures will have more than one fracture. Commonly, a fracture of the symphysis or parasymphysis region will

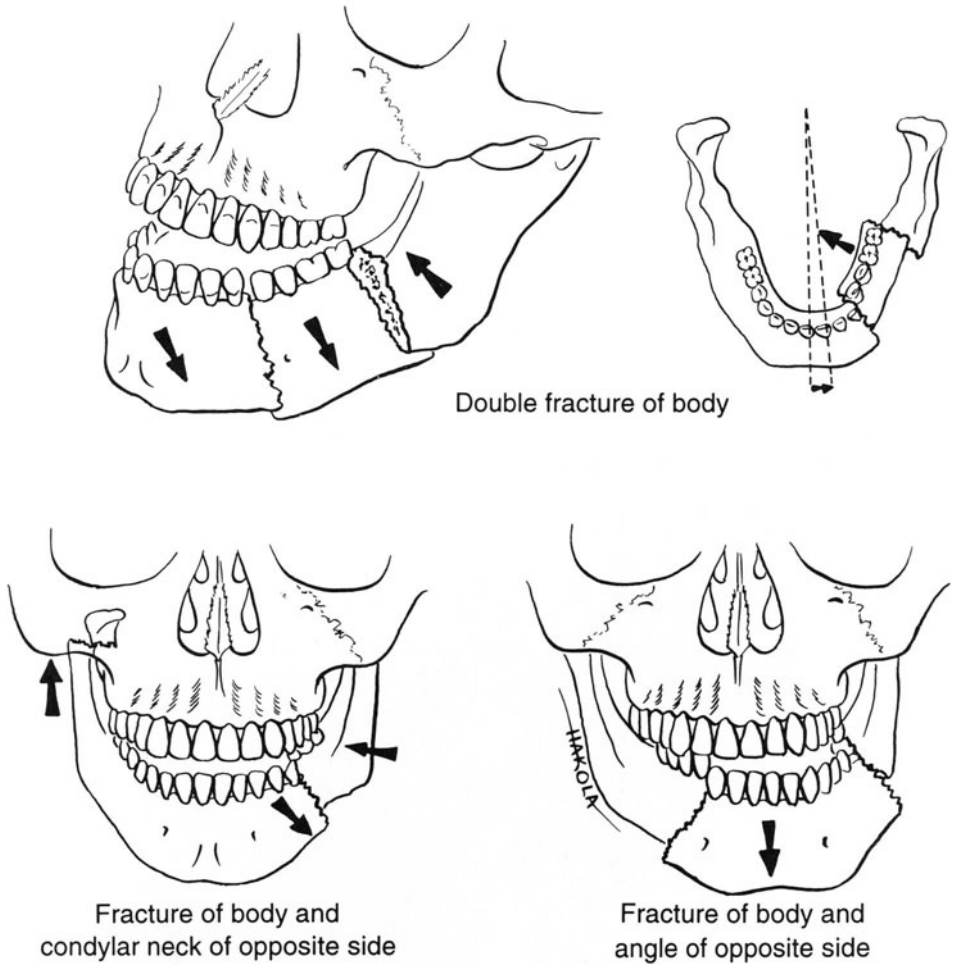

Figure 9.7. Body fractures.

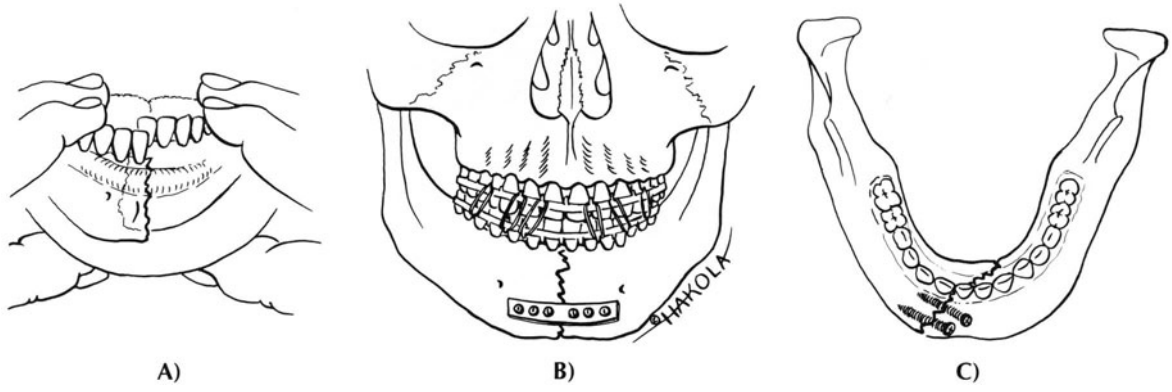

Figure 9.8. Symphysis and parasymphysis fractures. (A) Testing for mobility and displacement of fragments. (B) Plate fixation of a symphysis fracture. (C) Lag screw stabilization of a symphysis fracture.

produce a condylar or subcondylar fracture. When a severely comminuted fracture is present, the preinjury dental occlusion should be established by placing the patient into intermaxillary fixation with arch bars prior to reducing the multiple pieces with wires or microplates. A large mandibular plate is then applied. If a plating system is used for mandible fractures, one should be exceedingly careful in using plates in the compression mode. One should remember that the goal is to produce the patient's preinjury occlusion, and overzealous compression of fracture segments may be detrimental to obtaining good occlusion. If the fracture is severe enough to produce loss of bone, then immediate bone grafting and/or placement of a long reconstruction plate should be considered to span the defect and maintain space.

Teeth in the Line of Fracture

There are many instances in which teeth are involved in the line of fracture. This most often occurs in the mandible, particularly in the symphyseal and angle areas. Those fractures involving teeth in the line of fracture have to be considered as compound fractures, and as such, patients should probably be admitted to the hospital for parenteral antibiotic therapy. As a general rule, those teeth involved in the line of fracture should be extracted (Fig. 9.9). There is a great risk in retaining those teeth as they are prone to keep an open conduit from the oral cavity with its high bacterial count into the fracture site. The surgeon treating fractures of the mandible should not depend on retaining those teeth for the purpose of stabilization and/or maintaining space for ana-

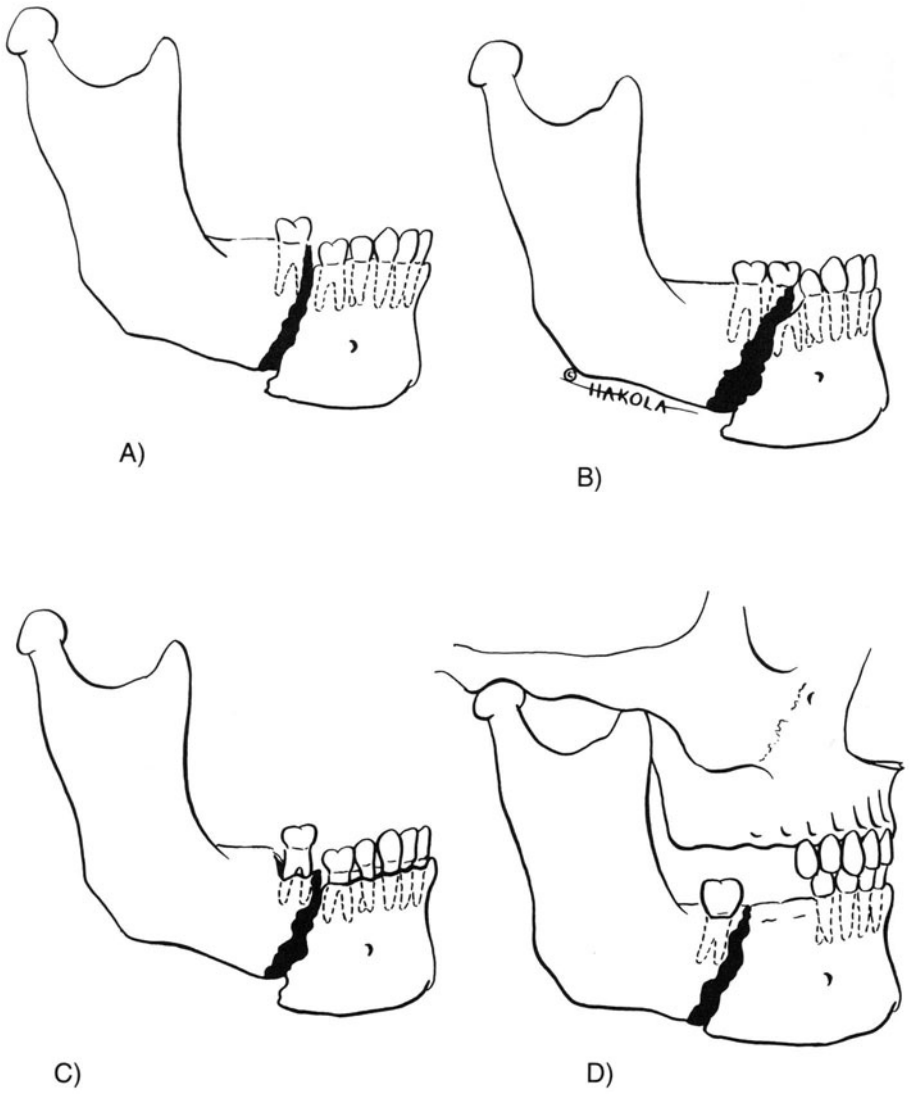

Figure 9.9. Indications for dental extraction of teeth in the line of fracture. (A) Displaced fracture containing a tooth. (B) Fracture of a tooth. (C) Severe peridontal disease. (D) Functionless tooth.

tomic reduction. The risk of osteomyelitis is very high. The decision to retain a tooth in the line of fracture must be made with the idea that there is a risk of inducing a chronic osteomyelitis at the fracture site. There are instances where teeth, primarily in the symphyseal area involving the mandibular anterior teeth, and also bony impacted posterior teeth (e.g., third molar), can be retained with a somewhat lower incidence of risk. The risk of osteomyelitis should be discussed with the patient prior to surgical intervention.

Fractures of the Edentulous Mandible

With the loss of teeth, there is resorption of the alveolar ridge or superior border of the mandible. A longstanding edentulous state may lead to mandibular heights of 1 cm or less, which of course is highly susceptible to fracture. Fractures that are stable or have minimal displacement may sometimes be treated by soft diet alone. With displacement or mobility, open reduction with internal rigid fixation should be the treatment of choice. When the height of the mandibular body is less than 10 mm, one should give consideration to primary bone grafting. Dental splints may be a useful adjunct in the treatment of edentulous fractures.

Pre- and Postoperative Management

The time for operative intervention of mandibular fractures should take place within 3–5 days after the injury, so that soft tissue scarring does not interfere with anatomic reduction and facial symmetry. Early reduction and stabilization not only initiates healing that much faster; it also reduces the intensity and duration of pain. In the event the patient's medical condition precludes surgery, the application of arch bars and intermaxillary fixation should be performed. One must remember that the goal of treatment of mandibular fractures is to reestablish the patient's preinjury occlusion. Therefore, it is imperative that the preinjury dental occlusion should be established and maintained with adequate intermaxillary fixation prior to the application of rigid internal fixation. It should be remembered that once the fracture has been reduced and rigid fixation applied, the fixation should be released intraoperatively to ensure proper occlusion. If the occlusion is not correct, the rigid fixation should be completely redone. If the operator feels that the stability of the rigid fixation is excellent, the patient may be taken out of occlusion at the end of the procedure. Subsequently, light elastic traction may be used for the first week or two to put the muscles at rest. When the operator feels less confident about the rigidity of internal fixation, a greater length of time may be required for intermaxillary fixation. One should not hesitate to leave the patient in intermaxillary fixation for 4–6 weeks to ensure proper bone healing. When intermaxillary fixation is being used, careful attention should be paid to good oral hygiene as well as diet. A high-calorie, high-protein diet of blenderized food as well as commercial supplements should be used. Physical therapy is an essential part of the post-IMF period, especially in condylar fractures as was previously discussed.

Complications

Major complications of mandibular fractures include infection, malocclusion (malunion), and nonunion. Patients are normally maintained on antibiotics for at least one week. With any sign of a wound infection, the area should be surgically explored and debrided. Infection may lead to nonunion of a fracture site. With the use of rigid fixation, the diagnosis of a nonunion may be more difficult. Traditionally, the nonunion has been observed with mobility at the fracture site, which may be precluded with the use of the rigid fixation. Therefore, persistent pain or a subsequent wound infection should alert one to the possibility of a nonunion. The lack of evidence of radiographic healing for a period greater than 3 months' time may suggest the possibility of a nonunion. The source of nonunion should be identified and eliminated, all necrotic tissue debrided, and then it must be determined whether additional internal fixation or external fixation is indicated. Subsequent bone grafting may be required.

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CHAPTER 10

Midface, Upper Face, and Panfacial Fractures

William A. Crawley and Henry C. Vasconez

Nasal Fractures

A nasal bone fracture is usually diagnosed from clinical signs and symptoms such as bruising, swelling, pain, epistaxis, and, intranasally, nasal airway obstruction with a deviated septum or septal hematoma. The nasal bones may be mobile or deviated laterally. X-rays, such as proper nasal bone films, Water's view, or, at times, a CT scan, are generally used to document a nasal fracture, but the definitive diagnosis is usually made by clinical examination.

Stranc and Robertson[1] have proposed a useful classification of nasal fractures based on frontal and lateral forces of impaction. Frontal impact nasal fractures may be divided into degrees of severity designated as Planes 1, 2, and 3: Plane 1 is a simple nasal bone fracture, Plane 3 is a nasoethmoidal orbital fracture, and Plane 2 falls between 1 and 3 (Fig. 10.1).

Treatment of a Plane 1 fracture involves reduction of the nasal bones and septum. This procedure requires the use of proper instruments, such as Asch forceps, and it can be performed in the emergency room while the patient is under sedation or local anesthesia. Treatment of the Plane 3 fracture, a nasoethmoidal-orbital fracture, will be discussed separately. A Plane 2 nasal fracture may require open reduction and internal stabilization, and, possibly, bone grafting.

Fractures of the Zygoma

The zygoma, or malar bone, forms a major buttress between the maxilla and the cranium and involves the orbit. On clinical examination, a fracture of the zygoma is revealed by periorbital ecchymosis and subconjunctival hemorrhage, paresthesia of the infraorbital nerve, depression of the malar bone, a step-off or tender point at the orbital rim, globe dystopia or enophthalmos, a hematoma of the buccal sulcus, and, occasionally, difficulty in chewing or trismus. A CT scan is the preferred method of radiographic evaluation because it is unequaled in its ability to define the walls of the boney orbit and zygomatic arch involved in zygomatic fractures. A coronal CT scan will provide the best view of the orbital floor component.

Treatment depends upon the degree of displacement and orbital disruption. No operative intervention may be required for nondisplaced or minimally displaced fractures without significant orbital disruption and with no abnormalities of the globe or visual disturbances. An open reduction should be performed when a greater degree of displacement and significant boney orbital disruption exist.

For many years, fractures of the zygoma were treated with an open reduction of the orbital rim and frontozygomatic suture. When this procedure was used, however, the downward pull of the masseter muscle created an axis of rotation that produced a postoperative deformity (Fig. 10.2). This deformity consisted of lateral and inferior displacement, flattening of the malar bone, enlargement of the entire orbit, and, consequently, enophthalmos. This condition was the result of an inadequate reduction and fixation. Correction is accomplished by anatomic open reduction and rigid fixation of the important zygomaticomaxillary buttress, in conjunction with the infraorbital rim and frontozygomatic fractures. Generally, a slight depression of the zygomatic arch, revealed by CT scan or clinical exam, can be elevated and held into position without an open reduction. Laterally displaced or comminuted fractures of the zygomatic arch will frequently require an open reduction via a coronal incision.

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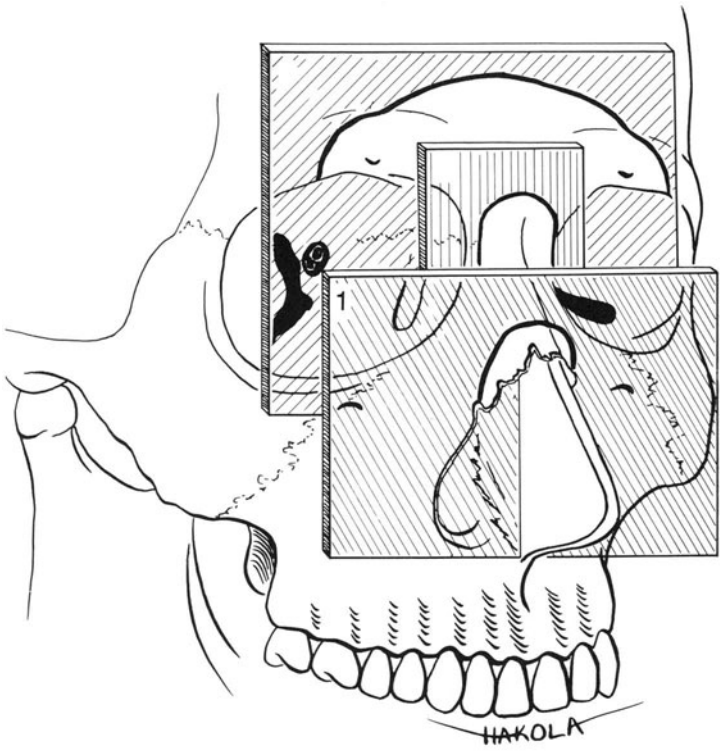

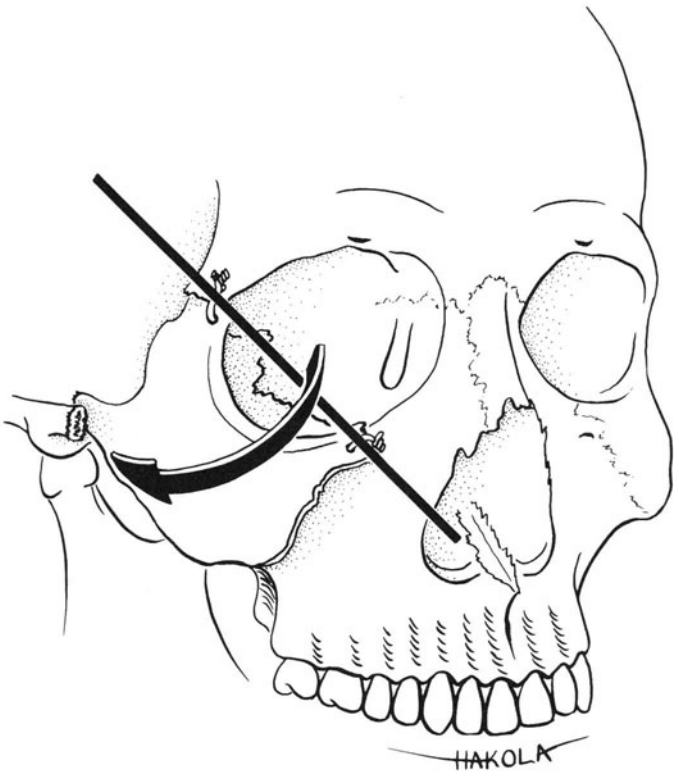

FIGURE 10.2. Axis of rotation with 2-point wire fixation.

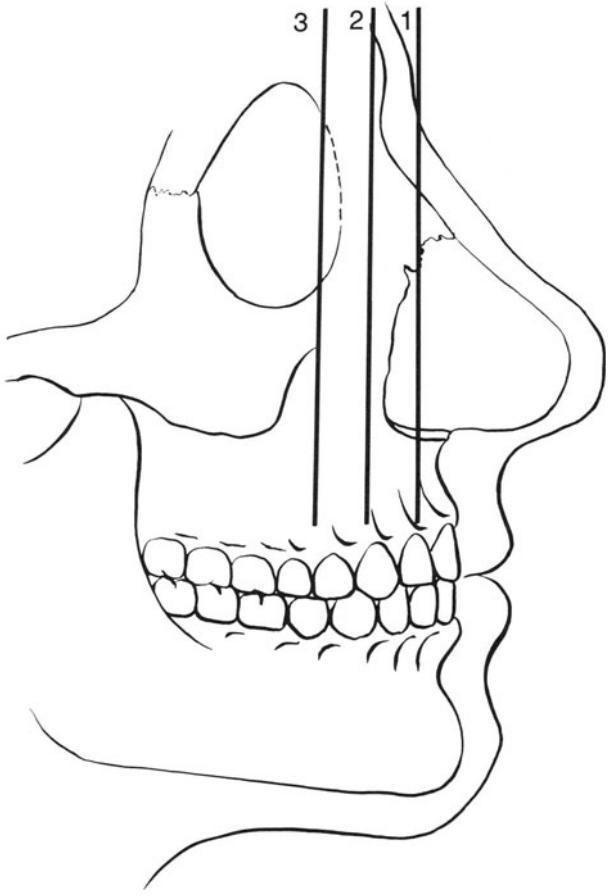

FIGURE 10.1. Planes of naso-orbital-ethmoid region.

The operative approach to open reduction and internal fixation of a zygomatic fracture consists of a transconjunctival incision or a lower eyelid subciliary incision, with a skin muscle flap and exploration of the orbital floor.[2] The single lower eyelid incision can be extended up and out; this incision has also been used to expose the frontozygomatic suture, taking down the lateral canthus in the process. Alternatively, the lateral extent of an upper eyelid blepharoplasty incision may be used for access to the frontozygomatic suture. Exposure of the zygomaticomaxillary buttress requires a gingivobuccal sulcus incision made 0.5 cm above the attached gingiva in the upper buccal mucosa.

Once complete exposure has been obtained, the fracture must be precisely reduced prior to fixation. When rigid fixation systems are used, two, three, or four points of fixation may be needed to achieve stability. Although the frontozygomatic suture is the strongest articulation, it is the least reliable indication of an adequate reduction. The single most reliable indicator of an adequate zygomatic reduction is proper alignment with the greater wing of the sphenoid, which should be made visible. Microplating systems are now most frequently used at the infraorbital rim, whereas miniplate fixation is used for the zygomaticomaxillary buttress. If a bone gap of greater than 0.5 cm is present in the buttress, a bone graft may be used. A large plate should not be placed on the front of the frontozygomatic suture because it will be easily palpable and may cause patient

dissatisfaction. Instead, either a plate on the posterior aspect of this articulation or a stainless steel wire in conjunction with plating of other articulations may be used. The zygomatic arch is essentially flat, not bowed, and, if it is opened, it should be reconstructed to recover proper anatomic contour. The floor of the orbit should always be explored. Treatment of the orbital portion of this fracture will be discussed under orbital fractures. Any soft tissue dissected from the zygoma should be refixed to the craniofacial skeleton. Refixation can be accomplished by careful fascial closure or by suspension of the superficial and deep fascia by means of nonabsorbable sutures through drill holes or around screws in the bone. Suspension will prevent future sagging and loss of normal contour.

Fractures of the Internal Orbit

Fractures of the internal orbit may involve not just the rim but also any of the walls of the orbit. Internal orbital fractures may be either blow-in or blow-out fractures, although the latter are by far the most common. The most frequently fractured portion of the internal orbit is the thin bone lying between the infraorbital nerve inferiorly and the ethmoidal vessels medially.

The most frequently seen physical signs of an orbital fracture are the combination of periorbital ecchymosis and a subconjunctival hematoma. Other signs or symptoms may include double vision, limited extraocular movement, and paresthesia of the infraorbital nerve. In addition to testing for visual acuity, the globe must be examined for injury. An ophthalmologic consultation should be strongly considered for any patient with a fracture of the internal orbit.

Radiographic evaluation is best performed by CT scan because such a scan can reveal all walls of the bony orbit, soft tissue herniation, muscle entrapment, and the anatomy of the optic canal.

The controversy concerning use of conservative (nonoperative) treatment or open reduction to treat fractures of the internal orbit has generated a large body of literature.[3] Generally, there are two major indications for an open reduction. The first is entrapment, evaluated by a forced-duction test. Initial muscle swelling that may subside after a few days may result in a positive forced-duction test; therefore, it may be appropriate to wait several days before a decision to operate is made. The second major indication for open reduction is orbital disruption, a condition that may produce enophthalmos or vertical dystopia (Fig. 10.3). This disruption is best

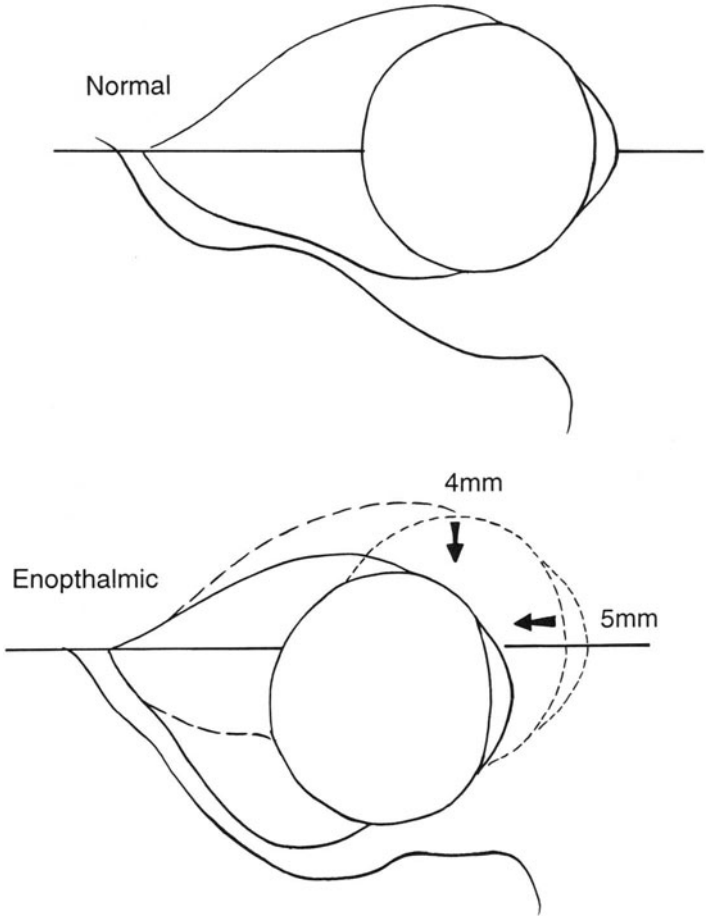

Figure 10.3. Mechanism of enopthalmos.

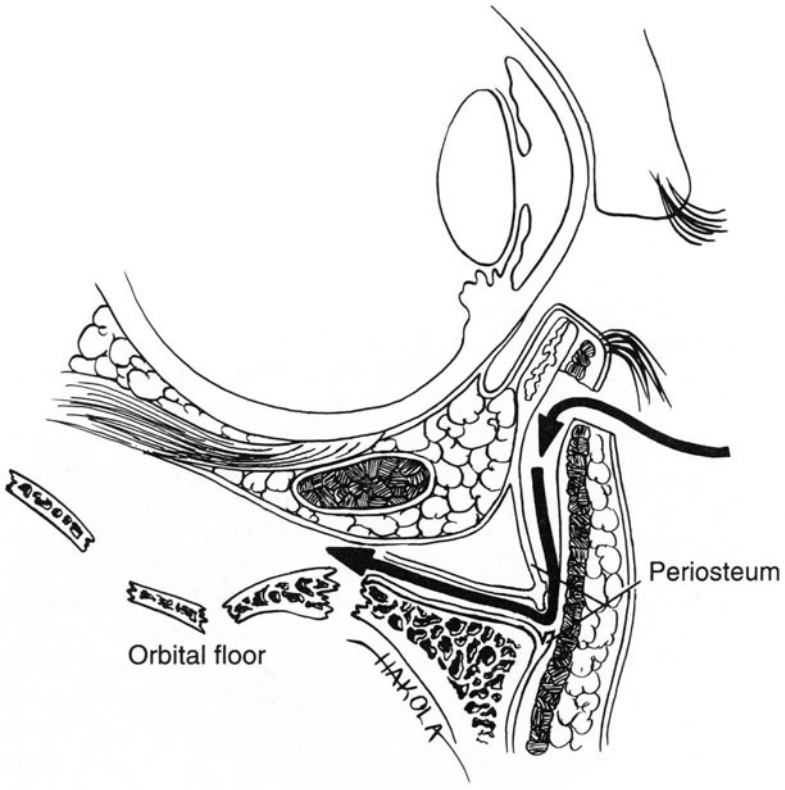

Figure 10.4. Skin muscle flap for exposure of orbital floor.

evaluated by CT scan. The exact size of orbital disruption at which surgery is required is not yet known. It is frequently said, however, that any defect greater than 2 cm should prompt an open reduction.[2]

The operative approach to an orbital floor fracture is usually via a subciliary incision with a skin muscle flap (Fig. 10.4) or a transconjunctival incision. Access to fractures of the medial orbit above the level of the ethmoidal vessels and of the roof will require a coronal incision. It is essential to identify all stable ledges of the defect. The goal of treatment is to reconstruct the normal bony anatomy of the internal orbit. The use of a dry skull may be helpful in planning. The orbital defect may be reconstructed with bone, an orbital plate, or a synthetic material. Autogenous bone is preferred. This bone may be taken from cranium, rib, or iliac bone.

LeFort (Maxillary) Fractures

The facial skeleton is composed of a series of horizontal and vertical buttresses. In the midface, the anterior buttresses consist of two nasomaxillary buttresses and two zygomaticomaxillary buttresses. The posterior buttress is the pterygomaxillary (Fig. 10.5).

At the turn of the century, Rene LeFort devised a system for classification of maxillary fractures (Fig. 10.6).[4] These fractures are usually bilateral. The LeFort I fracture extends from the area of the maxillary tuberosity to the nasal aperture; the LeFort II or pyramidal fracture extends from the maxillary tuberosity through the infraorbital rim and includes the nose; and the LeFort III fracture separates the middle and lower portion of the upper face from the cranium. These fractures may be seen in any combination.[5–11]

In addition to possible malocclusion, the symptoms of a maxillary fracture may include any of those associated with fractures of the zygoma, orbit, or nose. Except for a green-stick or immobile impacted fracture, the clinical finding is usually mobility of the involved segment. A conventional radiograph may be used as a scout film, but a more definitive diagnosis can be made with a CT scan. One of the major goals for treatment of Lefort fractures should be to reestablish the patient's preinjury dental occlusion. Therefore, the patient should always be placed in intermaxillary fixation when open reduction and internal fixation are being performed. The surgical approach for a LeFort I fracture may be through an upper gingivobuccal sulcus incision, exposing the four anterior vertical buttresses. For the LeFort II and LeFort III fractures, the subciliary and coronal incisions are best. At the LeFort I level, buttress plating is used (Fig. 10.7). Bone grafts should be considered for bone gaps

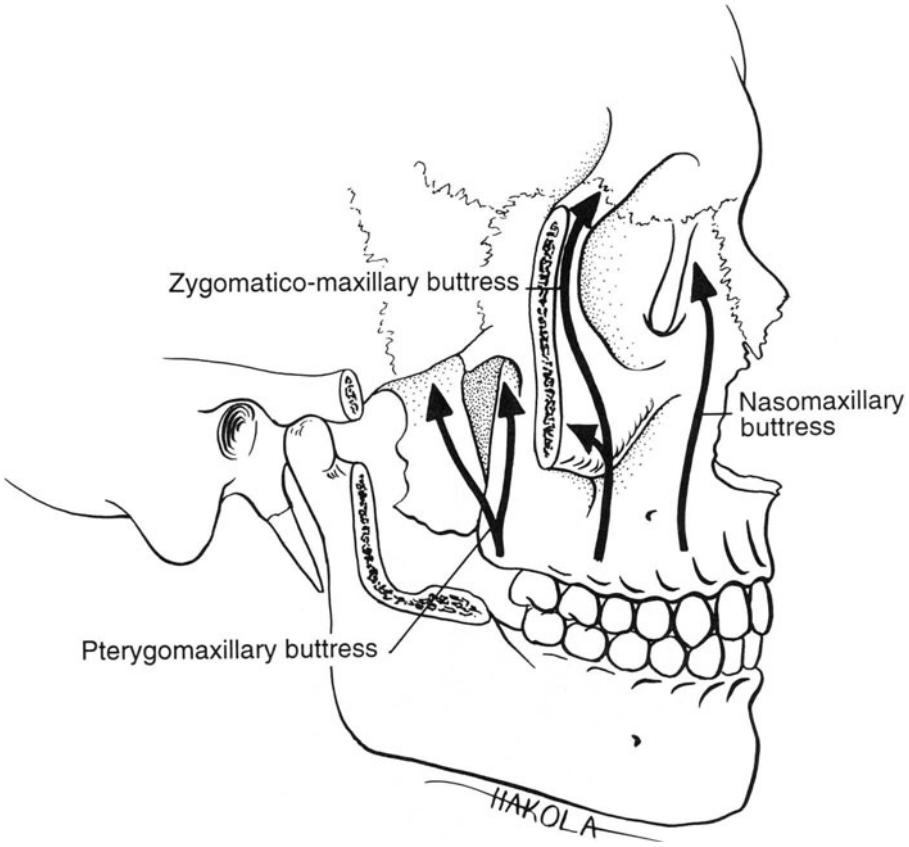

Figure 10.5. Vertical buttresses of the midface.

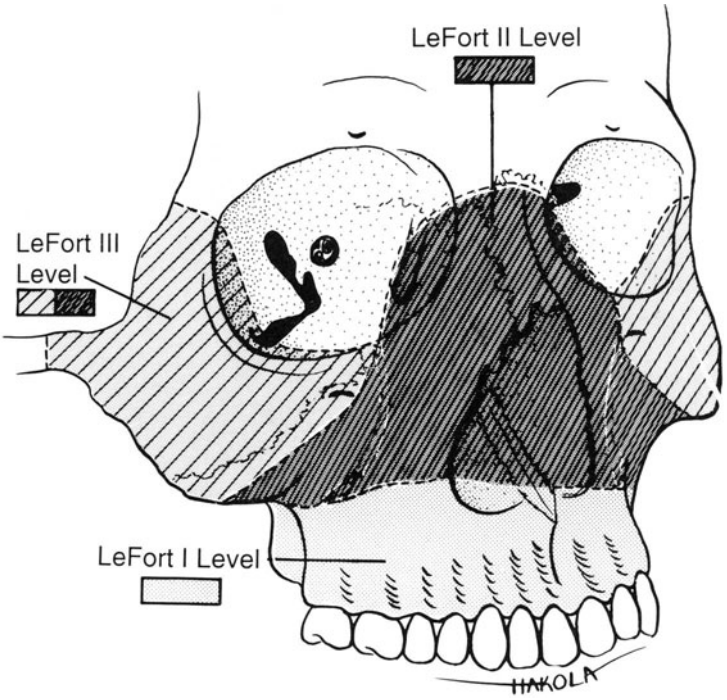

Figure 10.6. LeFort maxillary fractures.

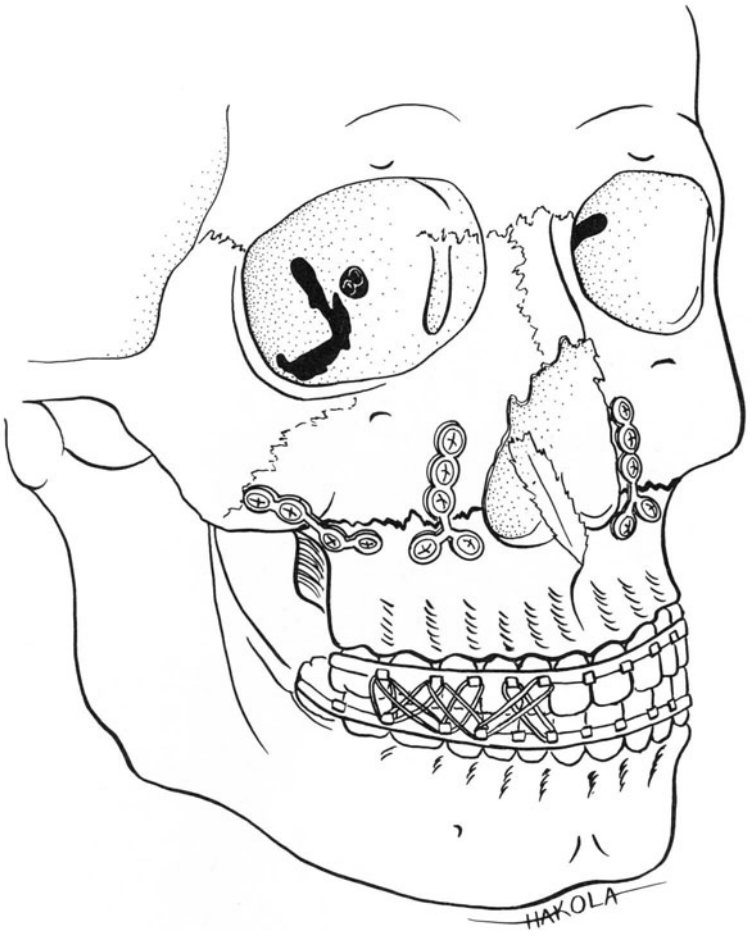

Figure 10.7. Plate fixation of LeFort I fracture.

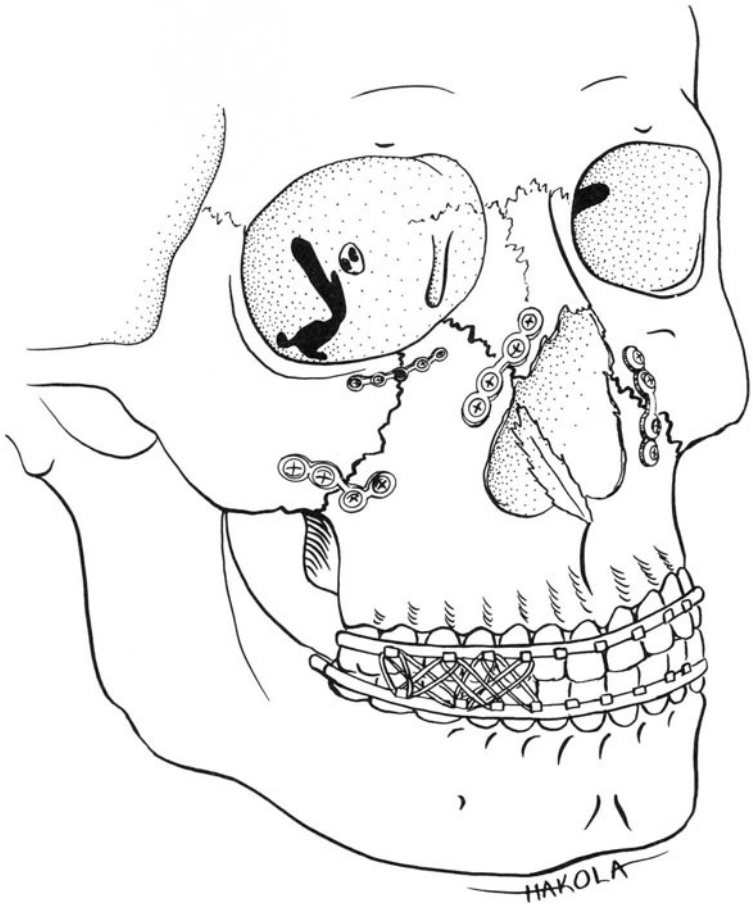

Figure 10.8. Plate fixation of LeFort II fracture.

greater than 0.5 cm. At the LeFort II and III levels, rigid fixation appropriate for the specific areas involved should be used (Fig. 10.8).

A second, but equally important goal in the treatment of LeFort maxillary fractures, should be to reestablish the patient's facial height and projection. Bone grafting may be necessary to accomplish this goal. Facial height is restored through anatomic reduction of the vertical buttresses of the face, whereas projection is a function of width. An anatomic reduction of the fractured facial bones with rigid internal fixation should be performed while the patient's teeth are in occlusion. Preinjury photographs may be helpful.

The patient with a green-stick or immobile LeFort fracture may present with malocclusion and a nonmobile maxilla. A CT scan confirms the diagnosis. Treatment consists of intermaxillary fixation. Occasionally, it may be necessary to complete the fracture and provide rigid fixation to reestablish the patient's preinjury occlusion.

Palatal Fractures

Sagittal fractures of the maxilla may produce a fracture through the hard palate. Clinically, one may see malocclusion with bruising or a laceration of the palatal mucosa. Once again, the CT scan is unequaled in confirming this diagnosis.

Traditionally, palatal fractures have been treated with dental splints to reduce the fracture and permit reestablishment of the patient's dental arch configuration. Recently, open reduction of the palatal fracture has been demonstrated to be helpful. This can be done from the oral, nasal, or anterior maxillary approach. If the oral approach is used, great care should be taken not to strip more mucosa than is absolutely necessary. Using the anterior maxillary approach, care should be taken to avoid the tooth roots. Reduction and fixation of the palatal fracture with a two- or four-hole mini- or microplate are especially helpful in adequately reestablishing the horizontal width of the maxilla (Fig. 10.9). One plate alone may not be sufficient; additional rigid fixation of the maxilla or, possibly, a palatal splint may be indicated.

Nasoethmoidal Orbital Fractures

In its simplest form, a fracture of the nasoethmoidal orbital area consists of a fracture through the medial orbital rim and the nose (Fig. 10.10). The potential for displacement of the medial canthal ligament is a major concern. Nasoethmoidal orbital fractures may be unilat-

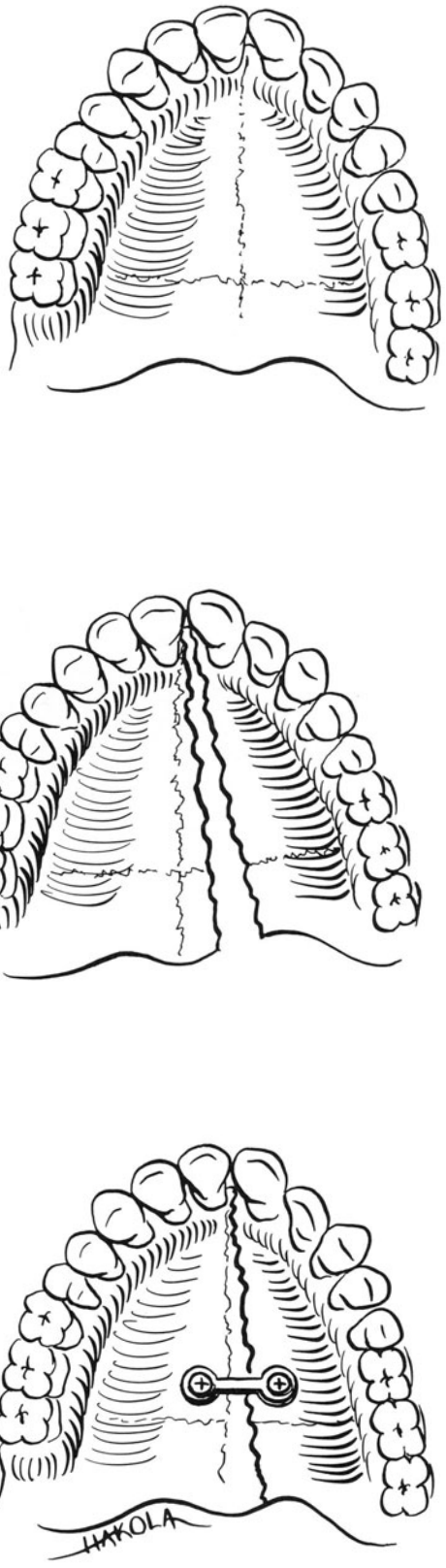

Figure 10.9. Fixation of palatal fracture.

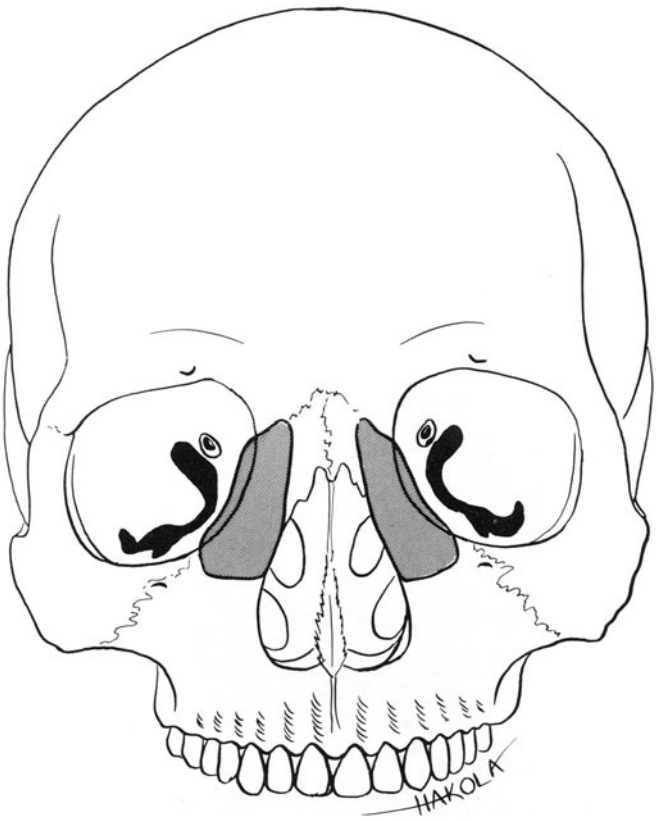

Figure 10.10. Naso-orbital-ethmoid injury.

eral or bilateral and may show varying degrees of displacement and comminution. Clinically, a nasoethmoidal orbital fracture should be suspected in anyone with a significant nasal fracture or a fracture of the medial orbital rim. Telecanthus, or widening of the intercanthal distance, suggests medial canthal disruption. One may also feel crepitation or movement over the area of the medial canthal tendon. A bimanual examination, in which a curved clamp is placed intranasally while the examiner's finger is placed over the bone surrounding the medial canthal tendon, may be used to check for mobility and displacement.

The radiographic evaluation must include a CT scan to reveal all associated fractures, presence of intracranial pathology, and status of the frontal sinus.

Patients with nasoethmoidal orbital fractures may experience cerebral spinal fluid (CSF) leak and injury to the nasolacrimal duct. Frequently, reduction of the fracture will be adequate for treatment of these problems. A persistent CSF leak may require lumbar decompression or, ultimately, a dural patch.

If there is an obvious open injury of the nasolacrimal apparatus, an attempt should be made at primary repair and stenting. In cases where no obvious injury can be defined, exploration may cause further damage and is discouraged. If a nasolacrimal duct obstruction should persist for 3–6 months, a dacryocystorhinostomy may be indicated.

Surgical treatment of the nasoethmoidal orbital injury is usually by means of a coronal approach. Proper anatomic reduction of the medial orbital rims and nasal bones may be accomplished with plate and screw fixation and the use of transnasal wiring. The medial canthal tendon should not be stripped from its bone. If, however, the medial canthal tendon is not attached to bone, a tendon-locking stitch should be placed with a nonabsorbable suture or wire, which can then be passed transnasally so that the tendon may be reduced anatomically.[12] Bone grafting may be needed in the medial or-

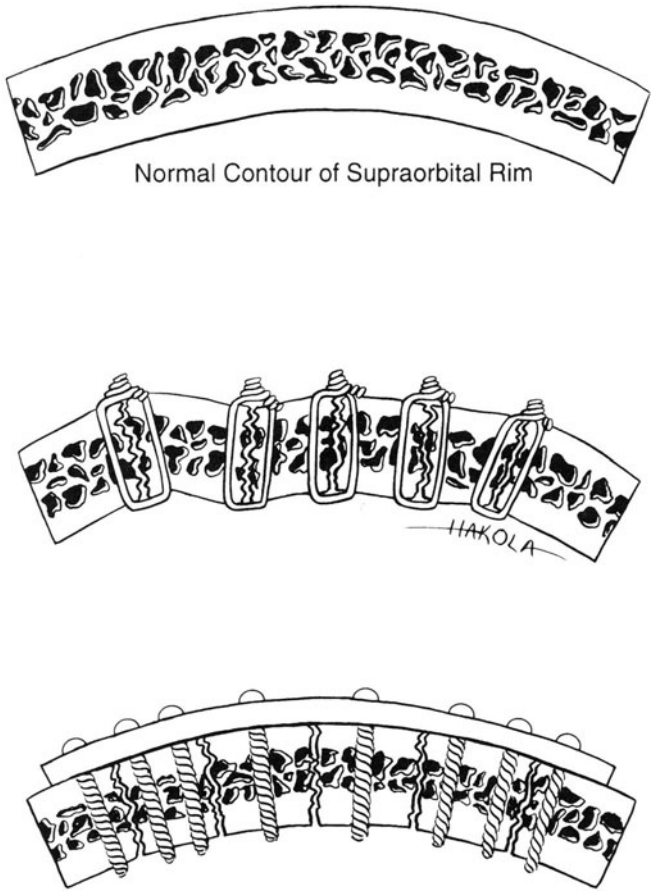

Figure 10.11. Wire versus rigid fixation of supraorbital rim.

bital rim and wall. Fractures of the frontal bone, which may be present, will be discussed subsequently.

Operative intervention within the first few hours after injury produces the best results for reestablishing the patient's nasomaxillary buttress and preinjury intraorbital distance. Early intervention also allows for reduction of the nose to preinjury dimensions and contour, frequently by use of a bone graft.

Fractures of the Frontal Bone

Fractures of the frontal bone may involve the frontal sinus in the central portion and the supraorbital rims in the lateral portion. These fractures may involve the base of the skull or the intracranial cavity.[13]

A bruise, laceration, deformity, supraorbital rim step-off, forehead paresthesia, ptosis, or downward and outward protrusion of the globe are signs of supraorbital fractures. The CT scan provides the best radiographic evaluation. Reestablishing normal convexity is best accomplished with plate and screw fixation, most commonly with microplates (Fig. 10.11).

Epistaxis, contusion, bruise, laceration, or depression of the glabellar area are indications of a frontal sinus fracture. A CSF leak may also be present. The CT scan provides an unequaled radiographic view. The anterior and posterior walls of the sinus can be evaluated on axial cuts. Coronal views can reveal the status of the sinus floor, orbital roof, and nasofrontal duct. Treatment of frontal sinus fractures has been a subject of controversy. Generally, depressed fractures of the anterior wall are treated with exposure and debridement of devitalized mucosal fragments, elevation and reduction of the bone segments, and rigid fixation. Fractures of the posterior wall are intracranial problems. In more severe fractures, the posterior wall is generally removed in a process called cranialization. Mucosa and bone fragments are debrided, the nasofrontal ducts are obliterated, a galeal-frontalis flap is rotated to provide a biologic barrier, and the anterior wall is reestablished or bone grafted if needed. In less severe fractures with injury to the nasofrontal duct, an assessment of patency is performed. This can be done with a dye such as fluorescein or a gentle probe. If widely patent, simple repair of the fracture is performed. If partial obstruction is encountered, a silicone stent can be placed for about 2 weeks to reestablish drainage. If occlusion of the nasofrontal duct is confirmed, treatment involves obliteration of the duct orifice by means of biologic tissue, commonly with chips of bone graft, debridement of all mucosa, and obliteration of the sinus cavity, preferably with bone graft or a vascularized flap (Fig. 10.12).

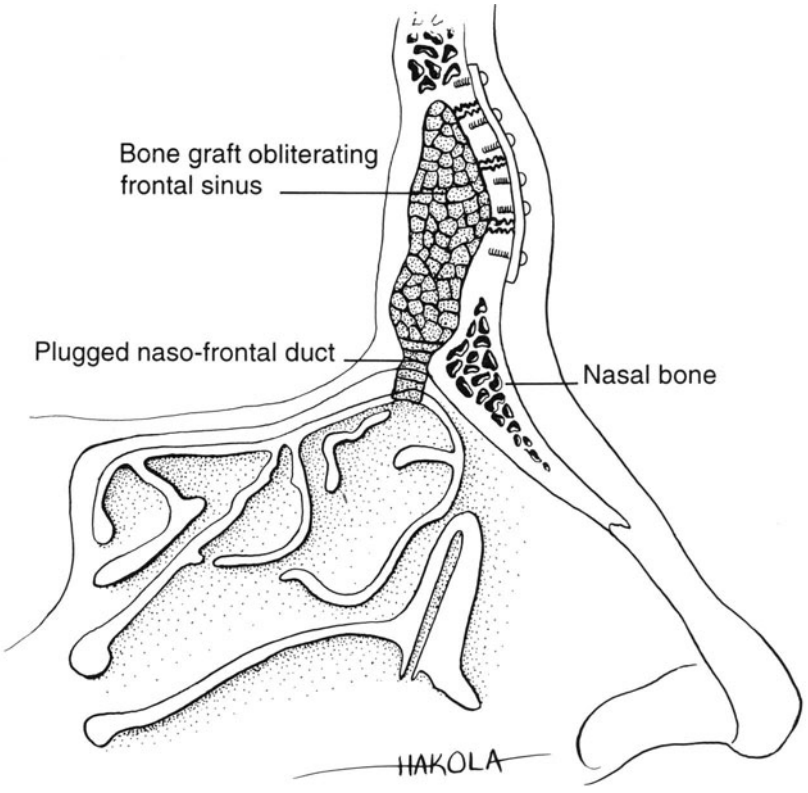

Figure 10.12. Frontal sinus obliteration.

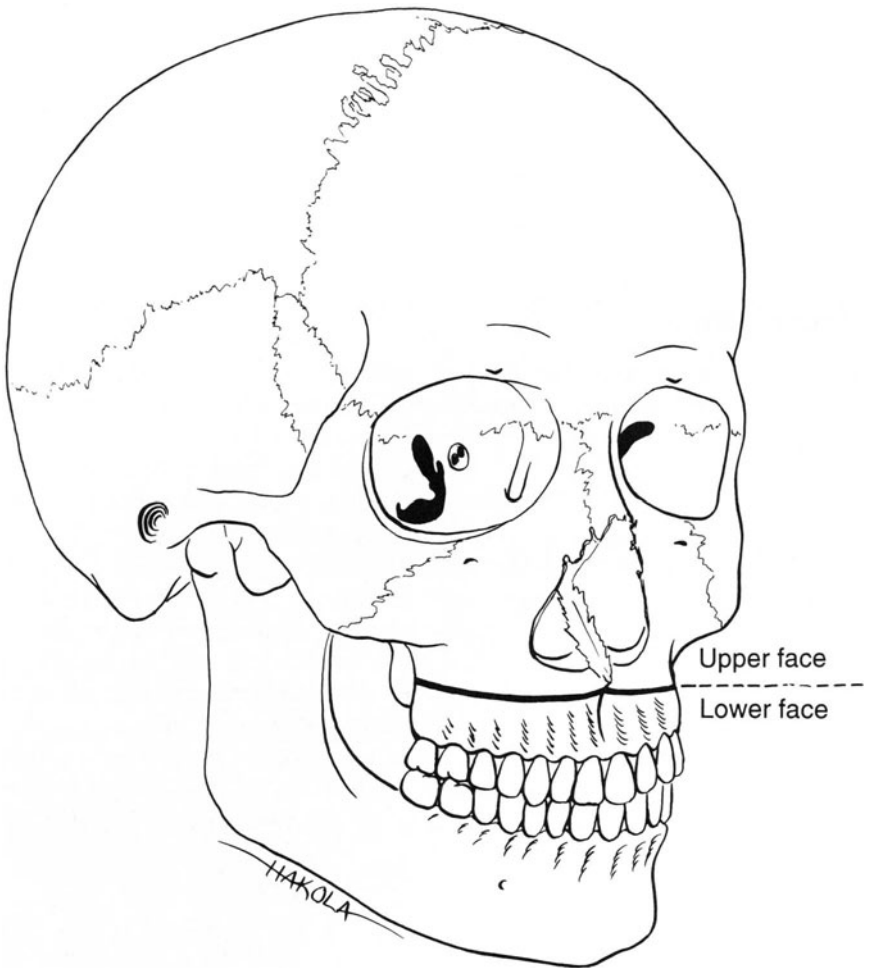

Figure 10.13. Division of upper and lower face at the LeFort I level.

Panfacial Fractures

Panfacial fractures are serious injuries to the upper and lower face. These fractures may involve all the areas previously discussed in addition to the mandible. The goals in treatment of these injuries are to reestablish the patient's premorbid functional status, including considerations of occlusion, facial proportions, and bone and soft-tissue healing. For the purpose of reduction and fixation, the face may be divided at the level of LeFort I (Fig. 10.13). In other words, reduction and fixation above and below the LeFort I level should be completed first; fixation at the LeFort I level should be the final step in treatment. The rationale for this approach is that the LeFort I level can tolerate imprecise reduction with fewer problems than can areas above or below that level.[14] Adequate exposure can be provided through several well-placed incisions. Five incisions are used for exposure of the anterior craniofacial skeleton: the coronal incision, with extension to the preauricular area; the subciliary incision; the upper gingivobuccal sulcus incision; the lower gingivobuccal sulcus incision; and the retromandibular incision (Fig. 10.14). After adequate dissection and correction of all fracture sites, the soft tissue fascia must be anchored to the craniofacial skeleton to avoid sagging or drooping. Replacement of the medial and lateral canthi to their proper position, resuspension of temporal muscle and fascia, and careful closure of mucosa and skin can avoid future defects that may be difficult or impossible to correct.[15,16]

The reciprocal relationship between facial width and projection is determined by the zygomatic arch (Fig. 10.15). As the width of the arch is decreased, the facial projection is increased. Facial height is a function of the vertical buttress of the facial skeleton previously discussed.

Once all fractures have been reduced and plated, intermaxillary fixation should be released and the occlusion checked with both condyles seated in the glenoid fossae. The occlusion obtained should be compared to the patient's dental models for accuracy. If the patient's occlusion is not correct, rigid fixation should be redone until proper occlusion has been obtained.

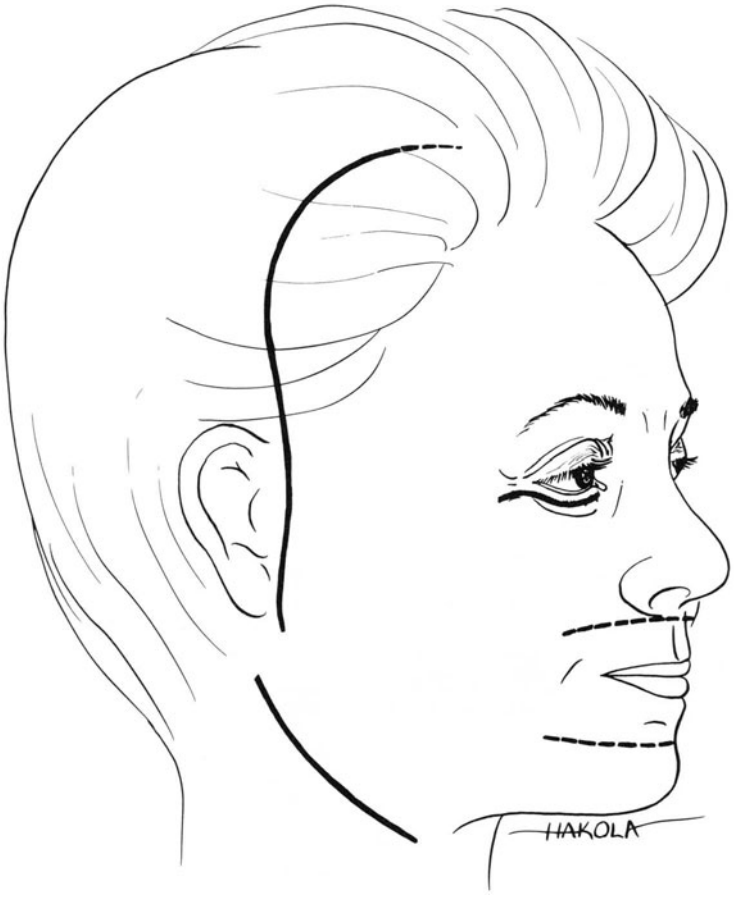

Figure 10.14. The five incisions of exposure of the craniofacial skeleton.

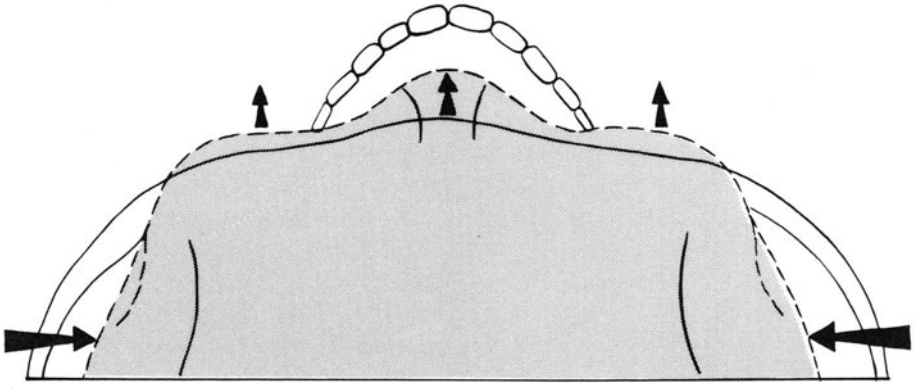

Figure 10.15. Reciprocal relationship of facial width to projection.

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CHAPTER 11

Facial Fractures in the Pediatric Patient

Jeffrey C. Posnick

Pediatric facial fractures are rare before 5 years of age. The incidence of facial bone fractures in the 1- to 5-year-old group has been reported to be 1.4% by Panagopoulos in 1947 and up to 4.8% by Rowe and Killey in their 1955 review of 55 fractures. MacLeannan noted that less than 1% of all facial bone fractures occur in children under 6 years of age. When they do occur, they are more likely to be "green stick" in nature, but this is not always the case. Posnick et al. have recently reviewed a series of craniomaxillofacial fractures occurring at a large pediatric referral center. The mechanism of surgery location and position of facial fractures, pattern of facial injuries, soft tissue injuries, and any associated injuries to other organ systems were recorded and fracture management and complications were recorded (Tables 11.1–11.5).

Many factors contribute to the higher degree of elasticity and resistance to injury of the pediatric maxillofacial skeleton before 5 years of age. Among them are the greater cancellous to cortical ration, the smaller head to face ratio, the rudimentary sinuses, the thicker more prominent soft tissues, and the shorter distance of falls when they do occur. All these factors give an inherent stability of the maxillofacial skeleton, consequently leaving the cranioorbital skeleton a more vulnerable area in this age group.

After 5 years of age the mandible and maxilla are filled with developing permanent dental follicles in addition to the erupted deciduous teeth. This along with the commencement of a more active school environment results in increased susceptibility of the facial skeleton.

Rowe and Kiley noted that in the 6- to 12-year-old group, the incidence of facial fractures was 4.4%. They indicated that with increasing age, the ratio of cranial to facial skeleton size decreased, resulting in greater prominence of the facial bones and higher incidence of facial injury (Fig. 11.1).

The greater osteogenic potential of the periosteum in children, with its rich vascularity, along with an increased metabolic rate, likely accounts for the rapid healing of fractured bony segments. It is for this reason that facial bone fractures in children must be diagnosed accurately and treated early after injury. In general, the basic principles for treatment of maxillofacial fractures in the adult applies to the child. A major difference relates to the mixed dentition phase (age 6 through 12 years), which presents challenges for fixation of dental arch bars, internal fixation and splints. When arch bars and splints are required innovative thinking is needed (Fig. 11.2).

During the mixed dentition, the maxillary and mandibular basal and alveolar bone are filled with developing permanent dental follicles in addition to the erupted deciduous teeth. This results in increased susceptibility of these portions of the jaws to fracture. Knowledge of the eruption patterns of the deciduous and permanent dentitions is needed to avoid injury to the teeth.

Emergency Room Assessment

Emergency treatment of children with maxillofacial injuries is the same as for all trauma patients. The initial priorities are for the provision of an adequate airway, control of breathing, control of hemorrhage, and prevention of aspiration.

The airway must be carefully examined and suctioned to eliminate any obstruction. The trachea and pharynx in a child is small and easily obstructed by blood or vomitus.

Endotracheal intubation is the preferred method to control breathing when necessary. The need for tracheostomy should be avoided if another means of maintaining the airway is available. In the young child, a

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Table 11.1. Mechanism of pediatric facial fracture by age category.

Age group (yr)	Traffic accident	Falls	Sports-related and altercations	Other
<3	1	9	0	2
3 to 5	12	8	4	1
6 to 12	32	12	9	4
13+	23	3	15	2
Total	68	32	28	9

From Posnick et al., 1993.

Table 11.2. Patient age and occurrence of pediatric fractures by region.

Age group (yr)	Cranium	Orbit	Zygoma	Midface	Mandible
<1	0	1	0	0	3
1 to 2	2	2	1	0	4
3 to 5	2	5	2	3	19
6 to 12	8	16	9	8	27
13+	4	17	9	12	22
Total	16	41	21	23	75

From Posnick et al., 1993.

Table 11.3. Pediatric fracture pattern by anatomic region and complexity.

Anatomic region	Subjects	No. of fractures	Fracture complexity		
			Group 1	Group 2	Group 3
Cranium	25	27	9	1	15
Orbit	41	73	7	5	29
Zygoma	21	22	4	0	17
Midface	23	31	3	0	21
Nose	17	23	6	4	7
Mandible	75	107	38	17	20
Dentoalveolar	32	44	8	11	13

From Posnick et al., 1993.
Fracture complexity resulting from trauma was represented by three groups: group 1, trauma involving a single fracture in a single anatomic region; group 2, trauma involving multiple fractures in a single anatomic region: and group 3, trauma involving multiple fractures in multiple anatomic regions.

Table 11.4. Management of acute pediatric fractures (n = 171).

No surgical treatment	Closed reduction (no. of fractures)	Open reduction (no. of fractures)
50	Reduction only (4) MMF (54) X-Fix (1)	Exploration only (13) Single fixation method (35) More than one fixation method (14)

From Posnick et al., 1993.
MMF, maxillomandibular fixation; X-Fix, external fixation.

Table 11.5. Age of pediatric patient with plated fractures.

Age group (yr)	No. plated fractures	Total no. fractures in group	Plated fractures (%)
3–5	8	37	22
6–12	18	83	22
13+	14	52	27

From Posnick et al., 1993.

tracheostomy may add an inordinate and late morbidity, and its weaning and removal can be a long-term process. One must also remember that endotracheal tubes for children are not cuffed and, therefore, not aspiration proof.

Finally, the amount of blood loss should not be underestimated in a child whose circulation becomes significantly compromised with less than a 20% blood volume loss.

X-Ray Studies

With reference to plain radiographs, the views that provide the most diagnostic maxillofacial information are the Waters View, Caldwell View, Towns View, and Panorex dental film. Right and left lateral oblique views of the jaws in the open mouth position provide added information about the condylar heads of the mandible. A focused CT scan provides the most meaningful infor-

Figure 11.1. Dry skulls of various ages we demonstrated in oblique view. The skulls are age approximately 6 months, 11 years, and 20 years.

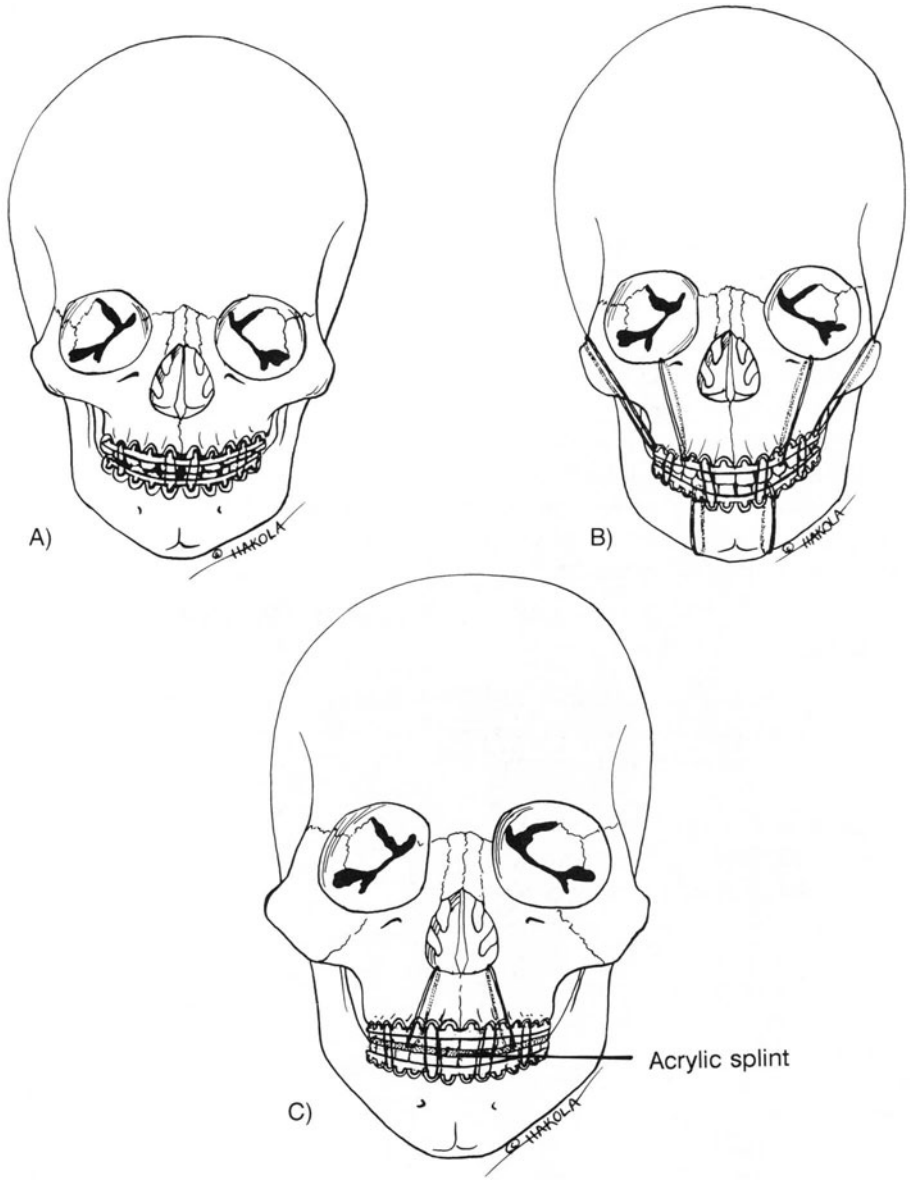

Figure 11.2. Drawing of three skulls at different ages. (A) At two years, (B) at six years, and (C) at twelve years. Different methods of wire fixation are shown: (A) shows arch bars and intermaxillary fixation only, (B) shows intermaxillary fixation with arch bars, circumzygomatic wires, infraorbital wires, and circumandibular wires, and (C) shows intermaxillary fixation with arch bars, an acrylic splint, and piriform sinus wires attached to acrylic splint.

mation regarding the facial skeleton. Both axial and coronal views are essential for a complete evaluation and can be obtained once the integrity of the C-spine is ascertained.

Associated Injuries

One must be on the alert for associated injuries to the cervical, thoracic, and lumbar vertebrae, as well as to the abdomen, chest, extremities, brain, and eyes. In the pediatric facial fracture series of McCoy, Chandler, and Crow, 41% of patients had associated injuries, other than the soft tissue facial wounds, with concussions in 31% and CSF rhinorrhea in 14%. Extremity fractures appear in 9.3% of all maxillofacial injuries in children, and closed chest trauma was apparent in 5.8%. Internal abdominal injuries appeared in 2.3% and blinding ocular injuries occurred in 3.6%. In the patients described by Kaban, et al., 29% of the patients had associated injuries other than facial wounds.

Signs and Symptoms

The clinical manifestations of fractures involving the mandible, maxilla, and the malar-orbital segments are similar for children as they are for adults. Pain and tenderness over any fractured facial bone with ecchymosis should arouse suspicion of a possible fracture. The pres-

ence of diplopia may be difficult to elicit in the very young child, but can be successfully tested in the older child (6 years and older), and may indicate an orbital floor or wall fracture. Inability to either open or close the mandible into satisfactory occlusion should also arouse suspicion of a fracture of the mandible, maxilla, or zygomatic complex.

Evaluation of the occlusion is a necessary part of the facial examination to determine whether there is any change from the preinjury function. The combined deciduous and erupting permanent dentitions (mixed dentition phase) may present complicated occlusal relationships. It is important that the preinjury occlusion and jaw relationships be considered prior to reduction and fixation of the fractured jaws. An understanding of normal dental facets and areas of erosion must be considered when moving boney fragments into proper mandibular and maxillary occlusal relationships.

Mandibular Fractures

Excluding nasal bone fractures, the most common facial bone fracture in the young child is the mandible. The majority of mandibular fractures occur in boys over 7 years of age.

Patients with mandibular fractures demonstrate at least some degree of malocclusion resulting from deviation and displacement of the fractured segments. There will also be pain on palpation at the fracture site, ecchymosis, and swelling.

Fractures involving the temporomandibular joint (TMJ) are accompanied by pain felt in the region of a condylar neck fracture with jaw movement or on direct palpation in the preauricular region. Bilateral condylar neck fractures will result in an anterior open bite malocclusion. This comes about by the upward pull from the masseter and pterygoid muscles with loss of posterior vertical height after medial forward displacement of the condylar segments out of the glenoid fossa by contraction of the external pterygoid muscles.

Deviation of the chin from the facial midline may occur with an isolated fracture through the mandibular angle without a contrecoup fracture on the opposite side of the lower jaw. These signs and symptoms of mandibular fractures can present clues to the location of the displaced segments. Careful overall examination and evaluation can give the correct diagnoses but the addition of radiographic imaging allows for more rapid and complete treatment planning.

The frequency of mandibular fractures by location varies with age. Mandibular fractures in children under the age of 6 years have frequent patterns of occurrences. These include (1) fracture of the body of the mandible. (2) fracture of the body of the mandible on one side, with a contrecoup fracture of the mandibular condylar process on the opposite side. (3) isolated unilateral fracture of the mandibular condylar process. (4) fracture of both mandibular condylar processes with or without a fracture through the symphysis region. (5) bilateral fractures of the body of the mandible. Another interesting statistic was noted by Lehman and Saddawi, who reported a 66% incidence of condylar fractures (when a mandibular fracture occurs) in children 11 to 15 years of age. In his study, children over 15 years of age who presented with mandibular fracture did so with the fracture occurring in the body and angle region 76% of the time.

Fractures in the young child frequently occur in the body of the mandible, which is packed full with partially erupted primary teeth and unerupted permanent dental follicles. When fractures occur adjacent to developing tooth buds, they rarely result in permanent injury.

Condylar Fractures

Condylar fractures in the child (or adult) can be classified as either *intracapsular* or *extracapsular* (Figs. 11.3 and 11.4).

1. *Intracapsular fractures* are often comminuted crush injuries involving the condylar head and its articular surface within an intact TMJ capsule. It also includes high condylar neck fractures within the TMJ capsule and attachments of the external pterygoid muscle on the condylar neck above the sigmoid notch. This muscle attachment produces medial and forward dislocation of the fractured condylar head–neck segment.
2. *Extracapsular fractures* are those below the TMJ joint capsule and extend from the level of the sigmoid notch downward and backward below the neck of the condyle to the posterior aspect of the ascending ramus.

Open reduction and internal fixation is rarely indicated for condylar fractures. Where a deciduous or mixed dentition is present, adequate treatment may involve immobilizing the jaws in occlusion to maintain vertical height of the ramus for a short period of time (approximately two weeks), followed by gradual mobilization with opening and closing exercises of the mandible. The role of the mandibular condyle as a growth center is not completely understood, but it does play an important role in facial growth. This conservative method of immobilization with a short period of intermaxillary fixation has stood the test of time, while other open reduction techniques have a limited role.

The TMJ joint is a ginglymodiarthroidal joint that differs from other bony joints in that articulating segments are not separated by hyaline cartilage covering the articular surfaces, but rather by an articular fibrous disc with only a small number of cartilage cells scattered throughout the disc. The articulation is also unique in that the basic movements permitted by the temporomandibular

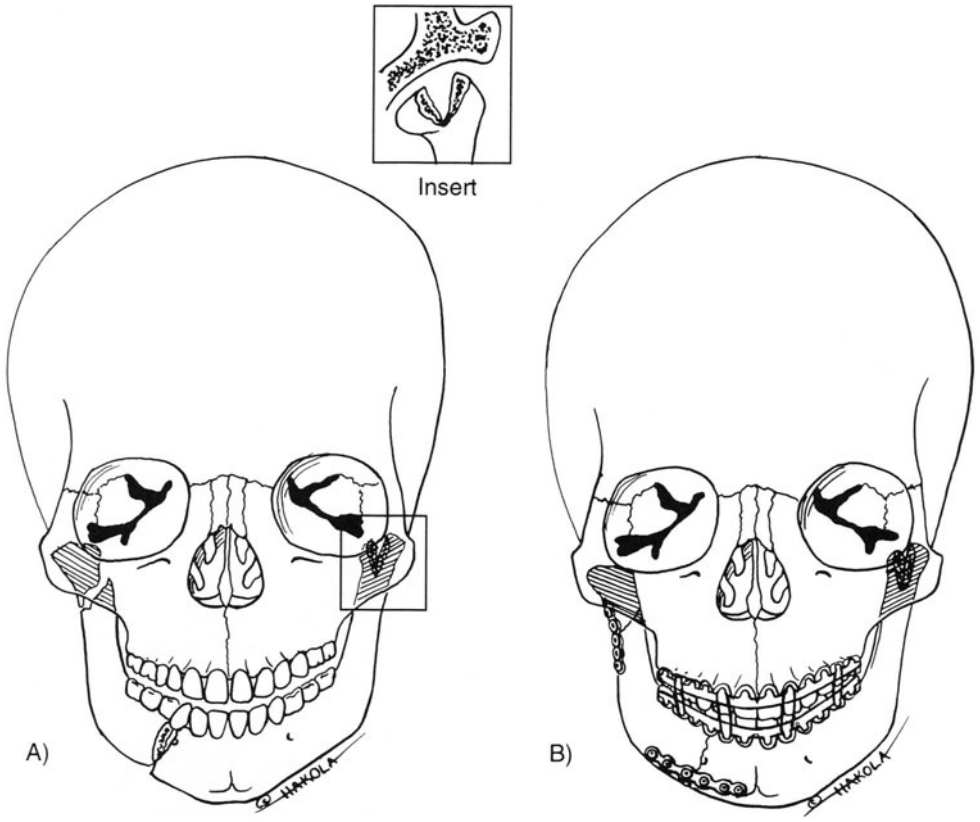

Figure 11.3. (A) Multiple facial fractures in an eleven-year-old child. They include a left intracapsular condylar fracture, a low right condylar neck fracture, and a right parasymphaseal fracture of the mandible. (B) Drawing showing postoperative right parasymphaseal plate and screw stabilization, right subcondylar open reduction with plate and screw fixation, and intermaxillary fixation with arch bars.

joint complex are (1) a hinge movement between the condylar head of the mandible and the articular disc, and (2) an anteroposterior gliding movement of a condylar head combined with the articular disc in the glenoid fossa of the temporal bone. Both right and left condyles are coupled and move as a unit, so that both TM joints pass through the same function at the same time unless the mandible is moving laterally (lateral excursion).

Body Fractures

When a fracture of the body of the mandible occurs, the muscle forces tending to reduce the fracture are generally favorable. This permits satisfactory reduction using closed techniques with IMF. In a very young patient with a green-stick fracture, a soft diet and limited activity alone may provide adequate treatment. If the fracture is badly displaced or if immobilization with IMF is to be avoided, then mini- or microplate and screw internal fixation at the inferior border of the mandible combined with application of an arch bar for stabilization at the dental level is preferred.

Parasymphyseal Fractures

When a parasymphyseal fracture occurs, displacement with widening at the inferior border and with a step off at the occlusal level (canine tooth region) will generally occur. Application of arch bars with intermaxillary fixation is often adequate treatment for an isolated parasymphyseal fracture, but may result in a step off between the lateral incisor and the canine tooth. If a contracoupe injury also occurs (i.e., contralateral condylar neck fracture) and an extended period of IMF is indicated, then internal fixation of the parasymphyseal fracture at the inferior border will stabilize the fracture to avoid the need for extended IMF. A small miniplate or microplate fixed with unicortical screws placed at the inferior border will limit the need for IMF.

Cranial Vault and Supraorbital Ridge Fractures

Fractures of the forehead and upper orbit are frequent craniomaxillofacial injuries in children less than 5 years of age. This part of the face is more prominent and

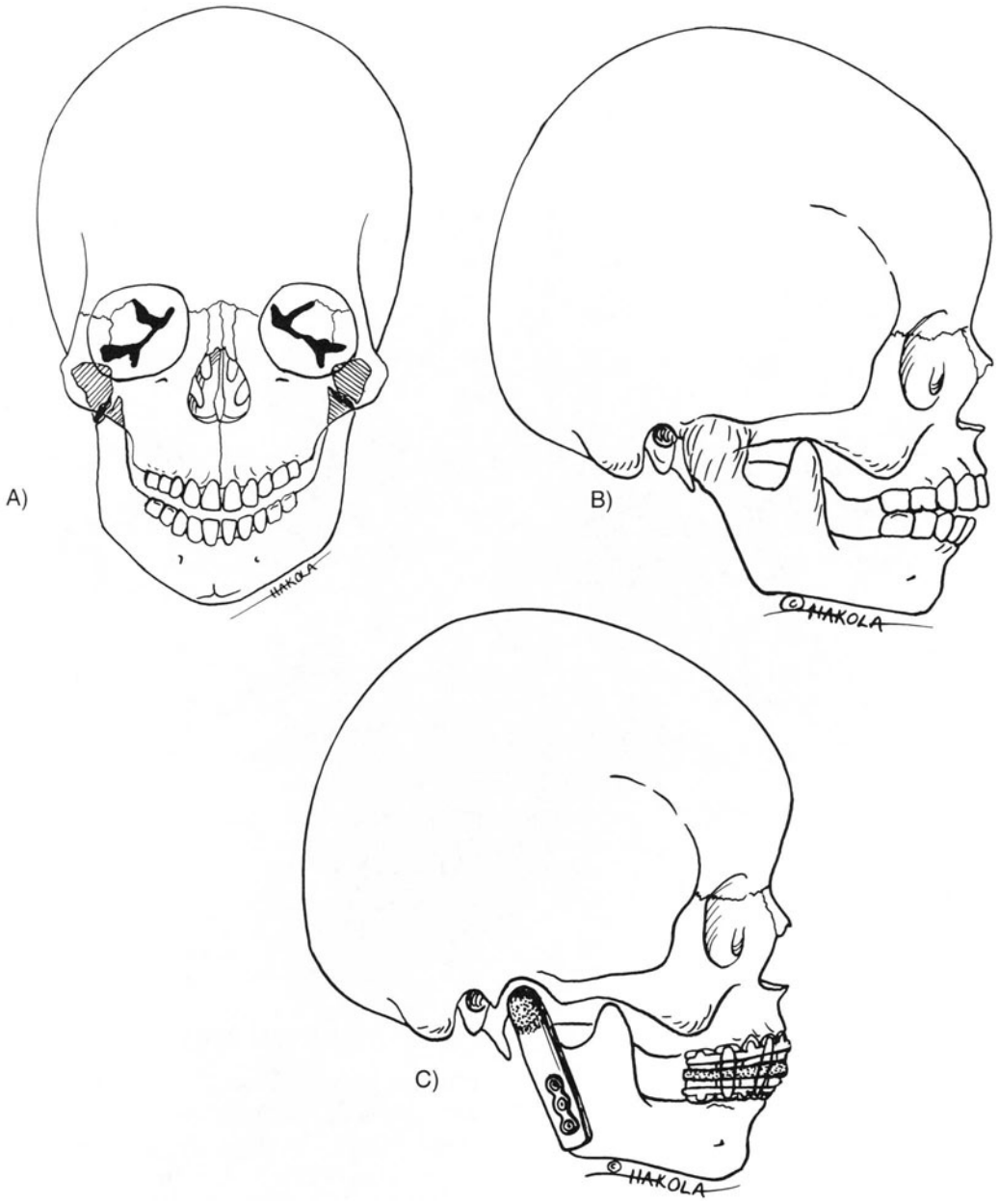

Figure 11.4. (A) A five-year-old child who presented with bilateral intracapsular condylar fractures of the mandible, which resulted in ankylosis of both temporomandibular joints. (B) Illustration of bilateral temporomandibular joint ankylosis to both zygomatic arches. (C) Postoperative reconstruction of temporomandibular joint with a costralcondral rib graft stabilized with a miniplate and screws. The patient is in intermaxillary fixation with arch bars and an acrylic splint.

exposed due to the limited development of the maxillofacial region.

When an injury in this region occurs, a complete craniofacial CT evaluation, neurosurgical assessment, and ophthalmologic consultation must be obtained.

When the fractures are displaced and require reduction, a coronal scalp incision is generally used. A combined procedure may be carried out, with the neurosurgeon conservatively debriding the injured brain tissue and repairing dural tears as necessary, while the plastic/craniofacial surgeon repositions the displaced forehead and orbital fractures. Fixation is achieved using either direct interosseous wires or microplates and screws as indicated. Autogenous cranial bone grafts may be required to reconstruct missing bone segments or orbital wall defects.

Nasofrontal Ethmoid Fractures

Fractures of the nasofrontal ethmoid complex can occur at any age. They generally result from direct trauma to the nasofrontal suture region. Preoperative assessment,

decisions about treatment, and incisions required for exposure and fracture reduction are similar for children as they are for adults.

When nasofrontal ethmoid fractures are associated with displacement, there is the need to anatomically reposition the bones. Exposure is generally gained through a coronal (scalp) incision. Fixation is achieved with microbone plates and screws. Boney defects managed with split autogenous cranial bone.

Management of the medial canthal ligament is similar to that in adults. If the medial canthus is attached to a bone fragment that is displaced, it is repositioned with the fragment without a separate medial canthopexy being required with the fragment.

Le Fort I, II, and III Fractures

Le Fort-midface fractures generally occur in older children and adolescents rather than in infancy or early adulthood (Fig. 11.5). When these injuries do occur, the same principles for treatment apply for children as for adults. It is important to restore the preinjury skeletal anatomy and then fix each fracture segment in place until boney union has occurred. Important differences relate to the upper jaw being smaller in children and filled with developing tooth follicles.

When exposure at the Le Fort I level is required, a maxillary circumvestibular mucosa incision gives excellent exposure to the fractured zygomatic buttress, anterior maxillary wall, and pyriform nasal aperture regions. When additional access to the zygomatic arches and supraorbital regions is required, a coronal scalp incision is added. If exploration to the orbital floor and medial orbital walls is needed, then either a subconjuctival, subciliary, or lower lid incision is used.

Fixation may be achieved with a combination of mini- and microbone plates and screws. Boney defects are reconstructed using split autogenous cranial bones, as in adults.

Zygomatic Complex Fractures

A complete zygomatic complex fracture is less likely to occur in a child than in an adult because of the incomplete development of the maxillary sinus. A green-stick fracture is more likely to occur.

The most common physical findings of a zygomatic complex fracture include periorbital ecchymosis, subconjunctival hemorrhage, and numbness of the skin overlying the zygomatic arch and within the infraorbital nerve distribution.

Indications for treatment are very similar to that in adults. That is, if the bone is displaced to avoid difficulties with late enophthalmus or the cosmetic problems of a flat cheekbone, then anatomical reduction with enough fixation to allow for boney union in the preinjury

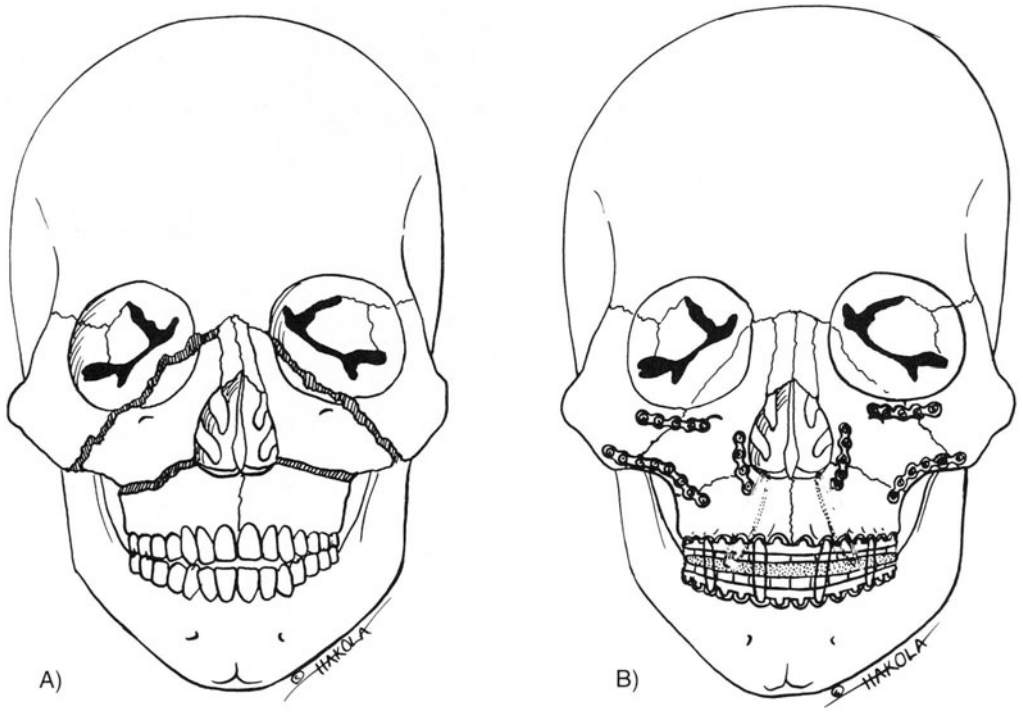

Figure 11.5. A fourteen-year-old adolescent with combined LeFort I and LeFort II fractures of the maxilla, associated with bilateral orbital floor fractures. (A) Illustrates the location of the fractures. (B) Shows reduction of the fractures with multiple plate and screw fixation. Intermaxillary fixation was accomplished with arch bars, an occlusal wafer splint, and bilateral piriform aperature drop wires attached to the splint.

position is required. Exploration of orbital floor defects with a need for reconstruction has similar indications to that of the adult.

Blow-Out Fractures

Blow-out fractures may occur as isolated injuries or be associated with a more complex set of fractures (i.e., zygomatic complex fracture, nasofrontal ethmoid, cranial vault-orbital roof, and Le Fort II or III fractures). A complete pediatric ophthalmologic assessment is mandatory. Radiographic imaging with coronal and axial CT scanning is essential for complete orbital evaluation.

Management of blow-in or blow-out fractures of the orbit are similar to those for adults. When intervention is deemed necessary, early treatment (within two weeks) is indicated, as these fractures heal rapidly in children and result in scar cicatration of the herniated orbital contents.

Nasal Fractures

In theory, trauma to the nasal septum during childhood could retard normal facial growth, resulting in a saddle nose deformity or marked midface deficiency with an angle Class III malocclusion. On the other hand, the nasal area is the most frequently fractured bone in the child's face and extensive midface growth retardation is rarely seen.

Fractures of the nasal bones in children should be treated in similar fashion to those in adults. Decisions should be made for or against conservative treatment of deforming fractures with late secondary reconstruction is not the preferred approach, open or closed reduction based on the extent of injury and expected outcome is the accepted approach. A septal hematoma after nasal trauma is said to be more common in children than adults. A hematoma should always be looked for and treated with drainage, to avoid septum necrosis or perforation.

Summary

There are differences in the craniomaxillofacial anatomy of children as compared with adults. Furthermore, these anatomical differences are constantly evolving during growth and development of the child. The resulting varied injury patterns that occur as the child grows to adulthood are predictable.

Complete and accurate diagnosis of all facial injuries are at least as important in children as in adults. The need for anatomic reduction and stable fixation to prevent malunion of the fractured bone is a key element in treatment.

After initial treatment of a facial fracture, the child's long-term follow-up by appropriate practitioners (maxillofacial surgeon and orthodontist) is essential to monitor facial growth and development. Late sequela may occur even when appropriate and prompt treatment is instituted. These are the consequences of growth restrictions and distortions secondary to the trauma event and the treatment rendered.

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CHAPTER 12

Facial Evaluation for Orthognathic Surgery

Bahman Guyuron and Steven R. Cohen

The comprehensive management of patients requiring orthognathic surgery to correct dentofacial deformities has developed rapidly during the last decade.[1] Technological advances in both orthodontics and orthognathic surgery have resulted in predictable treatment outcomes. The understanding that surgery compliments rather than eliminates the need for orthodontic therapy is an important consideration in planning comprehensive treatment for patients with dentofacial, as well as craniofacial abnormalities.[1] By a combination of orthodontic and surgical interaction, optimal dental and facial aesthetics, as well as masticatory function, should be achieved. The first step in formulating a definitive treatment plan for surgical correction of dentofacial, maxillofacial, and craniofacial abnormalities is an accurate evaluation of facial aesthetics.

Preoperative Evaluation

History

It is important to understand the patient's main reason for the consultation and what the patient perceives to be the problem. Not uncommonly, the patient believes the problem is different from what the surgeon concludes. A detailed assessment of the general medical condition, which is pertinent to the contemplated surgery, is done. This includes any history of cardiac or renal disease that would necessitate further consultation. A condition such as mitral valve prolapse will require prophylatic antibiotics treatment. A metabolic disease such as diabetes, unquestionably, would increase the incidence of infection and would require special consideration, should surgery be undertaken on this patient population. Immunocompromised patients should also be assessed carefully and surgical management should include a consultation with an appropriate specialist. These patients, generally, are not good candidates for major surgery and the rationale for surgery and medical necessity of the operation should be weighed very carefully against the potential and sometimes disastrous complications.

Hypotensive anesthesia should be considered for patients undergoing orthognathic surgery. This requires normal kidney function, so that the hypotension would not adversely affect the condition of the kidneys. Another important aspect of medical history is related to the coagulation system. Patients who bruise easily, or those who have bleeding tendencies, should be carefully investigated to rule out the possibility of some common, although mild, coagulation disorder (such as Von Willebrand's disease). Unless this is suspected and the bleeding time is checked, it might go undetected. Furthermore, all patients should be instructed to avoid aspirin-containing medications, or nonsteroidal anti-inflammatory medications, for at least 3 weeks prior to the surgery. Bleeding from the bone is heavily dependent on normal coagulation mechanism and any abnormalities of the coagulation system might result in serious complications.

Dental History

The past dental history is very pertinent. This includes any history related to periodontal disease, tooth extractions, and, more importantly, previous orthodontic work. Those who have been under prolonged orthodontic treatment might have evidence of root resorption, precluding further orthodontic work prior to orthognathic surgery. This must be carefully studied prior to a decision for orthognathic surgery. Those patients in whom dental compensation is currently being carried out by the orthodontist for what, in reality, is a skeletal problem must be noted. If such a condition is detected,

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compensations should be reversed (especially in the case of open bite) to reduce postoperative relapse. It is of utmost importance to have a recent full dental examination by a general dentist to rule out the possibility of cavities or undetected dental abnormalities.

Any history of temporomandibular joint pain or discomfort should be investigated carefully to discover any concomitant joint disease. Whether this is related to the skeletal abnormality or a totally unrelated condition, it should be clearly known prior to an attempt to surgically correct any dentofacial abnormalities. Some of the common symptoms that the patient might have, but might not volunteer, include clicking, temporomandibular joint pain, headaches (particularly in the area of the temporomandibular joint), and locking. This might require additional studies, such as magnetic resonance imaging, to rule out dislocation of the meniscus, and more importantly, to see if it is locked in a dislocated position or not.

History pertinent to the etiology of the dentofacial abnormality is also very significant. One has to look at the genetic predisposition as opposed to purely environmental factors. Siblings who exhibit similar dentofacial deformities might be very helpful in this regard. The patient should also be asked about any habits such as thumb-sucking or tongue thrust.

The patient in whom orthognathic surgery is contemplated must be questioned with regard to any speech problems that they might have had. Those patients with cleft lip and palate or other craniomaxillofacial anomalies who are destined to undergo maxillary or midfacial advancement are at risk of developing postoperative velopharyngeal insufficiency, and preoperative speech evaluation is necessary.[2]

Preoperative Psychological Assessment

Patients seeking changes in the basic skeletal structure of their face should understand as fully as possible that the changes may be dramatic. Profound psychological effects may result. It is, therefore, important that extensive discussion with the patient be carried out prior to the operation. A word of caution must be echoed. Changing the position of the dentofacial or craniofacial skeleton significantly affects a person's appearance. Alterations of body image are not always easily accepted, even when change brings improvement.[3] The young generally do well, but adaptability diminishes with age. Any change is thus accepted with greater resistance.

When contemplating orthognathic surgery, great care should be given *not* to compromise dental display. The show of maxillary incisors is associated with youth and should be preserved. To minimize postoperative psychological problems, patients should be counseled at length about the forthcoming changes in appearance. Although patients may look better and be "cephalometrically correct," the necessary psychological adjustment can be extremely difficult. It is not always easy to detect those individuals who experience untoward psychological sequela; however, preoperative counseling should be considered when doubt exists. Society's standard of facial beauty may be at variance with that of the surgeon, as well as with the patient's standard. Ultimately, treatment options and expected outcomes must be centered around the patient's own desires. Although anatomic and morphological change following surgery can be reasonably predicted, the psychological change the patient must undergo is far more unpredictable.

Clinical Evaluation—Anatomic Assessment of Facial Aesthetics

A systematic approach to the development of a comprehensive and individualized database requires a careful clinical examination. The exam must not only concentrate on occlusion, but equally on the aesthetic relationship of the craniofacial, skeletal, and soft tissue parts.

It can not be stressed enough that however detailed the surgeon's and orthodontist's clinical evaluation may be, it is the patient's concerns that must take paramount importance. A lack of understanding by the professionals involved in the care of any particular patient with dentofacial or craniofacial abnormalities may have serious repercussions on the outcome of treatment.[1]

A general clinical facial evaluation should include observation of the patient's skin in terms of thickness, color, and consistency. Any previous scars in the facial region should be observed. Patients with a light complexion and blue eyes, red hair, and freckles are notorious for formation of hypertrophic scars following any external incisions.

Frontal Analysis

Full facial evaluation proceeds from a general assessment of balance among various facial features to an assessment of specific relationships among them (Figs. 12.1 and 12.2). To avoid missing any critical abnormalities of the face, it is best to divide the face into three different zones—the upper, middle, and lower.

The upper zone will extend from the hairline to the eyebrows, the middle from the eyebrows to the subnasale (the junction of the nose and the lip), and the third zone will be from the subnasale to the menton (the lower-most portion of the chin). The facial divisions are then examined individually on frontal view and profile.

On the *frontal view* of the upper division, one has to check the forehead for the position of the hairline, the length and width of the forehead, and any asymmetry or irregularities. The eyebrows are then checked for

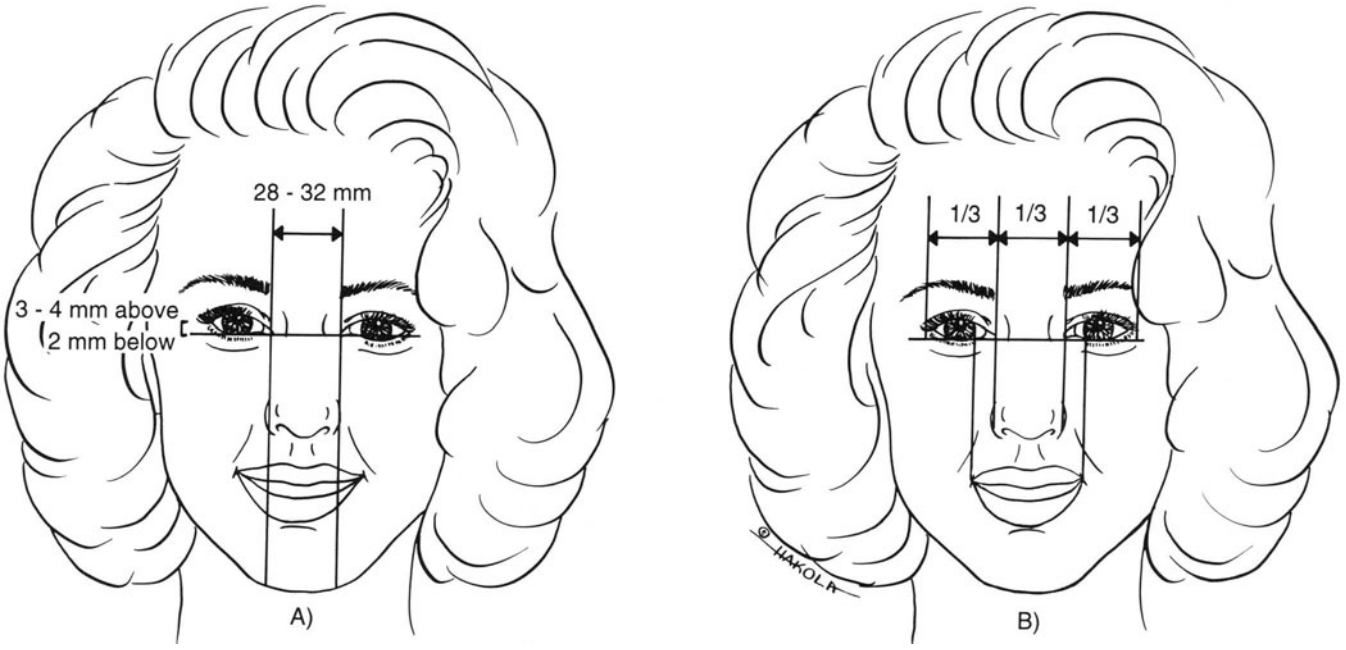

Figure 12.1. Frontal view demonstrating intercanthal distance and vertical location of the lateral canthi, and the relationship to canthi to oral commissures.

symmetry, direction, and position in relation to the eyelashes and hairline. The eyebrows are usually arched laterally, the highest peak of the arch being at the level of the lateral limbus. The lateral ends of the eyebrows are usually located cephalad to the medial ends. Reversal of this relationship will unquestionably disturb the facial balance and give the patient a sad appearance.

When examining the middle division from frontal view, the eyes are checked for symmetry in terms of size, position in the cephalocaudal, and medial and lat-

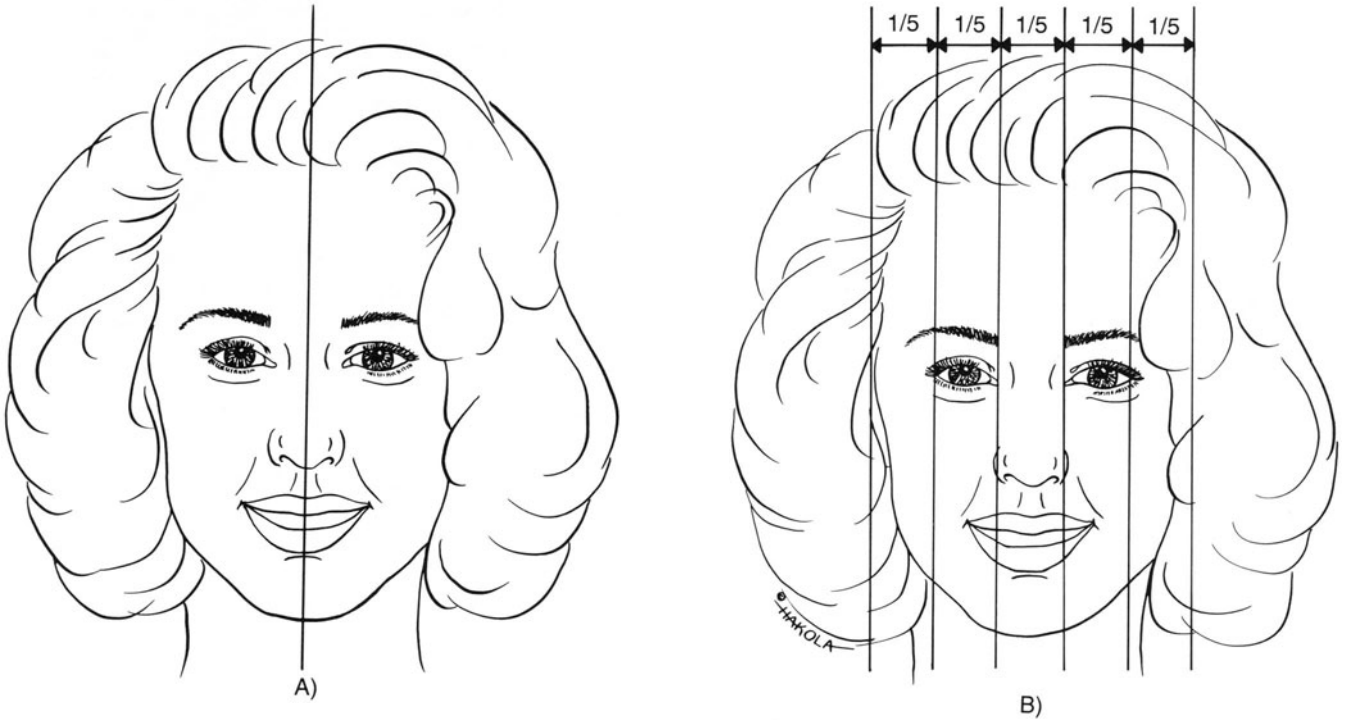

Figure 12.2. Evaluation of facial symmetry. (A) Right to left symmetry is checked by holding an object so it bissects the glabella, nasal tip, upper lip, and chin. (B) The face is divided approximately into fifths.

eral direction. Generally, the eye fissure follows the pattern of the eyebrow, meaning that the lateral portion of the fissure is further cephalad than the medial, creating a desirable angle of approximately 2 degrees. Significant deviation from this, such as lateral canthal displacement caudally in patients with Treacher Collins syndrome, or cephalad in patients with Down's syndrome, will disturb the facial balance and be perceived as a stigmata of the specific facial deformity. The intercanthal distance and interpupilary distance are also measured. The normal intercanthal distance is approximately 28–32 mm, which is equal to the orbital fissure distance from medial to lateral canthus (Fig. 12.1).[4,5] Reduction in intercanthal distance usually is a reflection of hypotelorism, whereas an increase in this distance may represent hypertelorbitism. However, this can only be concluded if the interpupilary distance is also increased. If the interpupilary distance is normal, yet the intercanthal distance is more than it should be, then the patient has telecanthus rather than hypertelorbitism.

The eyelids are then checked for movement and position. The upper eyelids usually overlap the iris by approximately 1–2 mm. Significant deviation from this would be an indication of lid lag, which denotes too much opening of the eye, or ptosis, which is significant overlapping of the iris by the upper eyelid. The presence of a lid cleft or absence of eyelashes should be observed.

At this point, the nose is examined. Each nasal unit should be checked: for example, the nasal bridge is noted for its width and direction, and the nasal tip is reviewed for its highlights, degree of prominence, and width of the alar domes and lower lateral cartilages. The alar base and nostrils are reviewed for width and symmetry.

In the middle division, the ears are also checked for prominence, visibility, location, direction, and symmetry. Again, any deviation from the normal standards will be noted. Generally, the antihelix is slightly visible on the front view and the helix is positioned almost parallel to the head, projecting about 1–1.5 cm off the mastoid bone. The length of the ear approximately equals the length of the nose. The lower border of the earlobe is at the level of the subnasale. In syndromal, maxillofacial deformities, the ears may have an abnormal position. The long axis of the ear creates a posterior angulation of approximately 30 degrees with the body axis.

Attention is then directed to the lower division. Upper lip length is first noted (Fig. 12.3). This is normally one half of the length of the nose and approximately one half of the distance from the stomion (junction of the

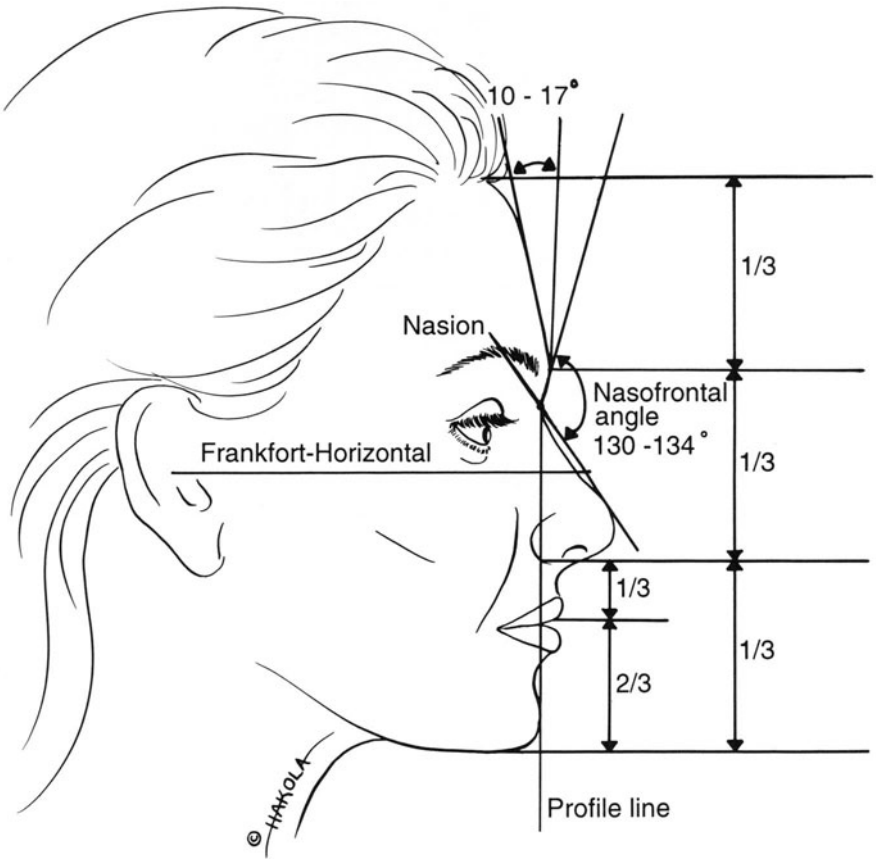

FIGURE 12.3. Profile of face demonstrating vertical and sagittal relationships.

upper and lower lip) to the menton. A short or long upper lip might be a reflection of an abnormality in the underlying skeleton. The upper lip should also be checked for the configuration of the philtral dimple and definition of the Cupid's bow. The oral commissure is then examined in terms of direction, which should be parallel to the floor and to an imaginary line connecting the medial canthi or the lateral canthi. In general, in the presence of normal facial muscle function, any tilt in the direction of oral commissure is an indication of skeletal abnormality. The oral fissure width matches the distance from one modial limbus to the other side. Major excess to this width is called macrostomia, which can be unilateral or bilateral, and decreased oral fissure width is termed microstomia. It is particularly important that the chin is checked for position, symmetry, and shape. An imaginary line connecting the midbridge to the philtral dimple and chin should be symmetrically located in the middle of the face.[6] Any deviation in this direction, again, is usually an indication of a skeletal deviation. In this view, also, the symmetry of the mandibular angle and its definition should be examined. Lastly, the submental area is checked for the presence or absence of excess skin, fat, or prominence of submaxillary glands.

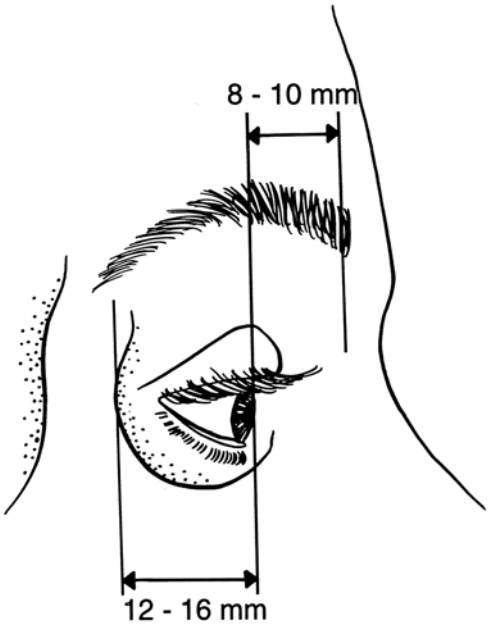

FIGURE 12.4 Close-up of profile view demonstrating relationship of anterior cornea to supraorbital ridge and lateral orbital rim.

Profile Analysis

At this point, the patient is asked to turn the face, so the true profile can be examined. In this view, the face is again divided into three divisions. The forehead prominence and presence or absence of frontal bossing is checked. Generally, the length of the forehead equals the length of the midface and lower face. A short forehead will disturb the balance of the face, as will a long forehead.

In viewing the profile, the morphologic appearance of the supraorbital ridges and lateral orbital rims as they relate to the anterior surface of the cornea is important. When the most anterior surface of the cornea is used as a reference, the normal supraorbital ridges project 8–10 mm anteriorly, while the lateral orbital rims sit 12–16 mm posteriorly[7] (Fig. 12.4). Forehead inclination, width, globe-orbital relationships, and the location of the eyebrows to the underlying supraorbital ridge should all be taken into account during clinical evaluation. The mean normal nasofrontal angle in men is 130 degrees and 134 degrees in women.[7] The female forehead is slightly less inclined than that of the male. A greater prominence of the supraorbital rim, especially at the glabella, is considered characteristic of the masculine face. Pneumatization of the frontal sinus begins at approximately age 7 years and varies in amount.[7] Hyperpneumatization can account for prominent glabellae in both sexes, resulting in a protruding lower forehead and accentuation of the nasofrontal angle.

The nose is examined for the angle between the forehead and the nose, or the nasofrontal angle, the presence or absence of a hump, and the degree of projection of the nasal bridge, which is usually 34 degrees for a female and 36 degrees for a male. Tip projection is checked, as well as the nasolabial angle, which is approximately 100 degrees for a male and 105–108 degrees for a female. The columella is projected caudal to the alar rim by 4 mm. The nasal spine position is checked. The normal position of the nasal spine is extremely important in assessing the relationship between the upper jaw and the nose. Although, occasionally, there is a truly large or small nasal spine, the overwhelming majority of patients have the appearance of nasal spine excess or deficiency as a reflection of excess or deficiency of the maxilla.

At this point, the lower division is examined on profile. Again, the length of the lip, projection of the upper lip in relation to the lower lip, labiomental groove, and chin projection are examined. Normally, the upper lip is located either at the level of the lower lip or slightly anterior to it. Reversal of this relationship may be an indication of a maxillary deficiency or mandibular protrusion (prognathia). However, there are rare situations when the deficiency is in the soft tissue itself, such as in the patient with a cleft lip deformity. It is important to distinguish such a lip deformity, which could be easily corrected using an Abbe flap without having to resort to orthognathic surgery.

A significant discrepancy between the upper and lower lip in the direction of the deficiency in the lower lip

projection, especially in the presence of an exaggerated labiomental groove, is an indication of retrognathia. The labiomental groove is generally about 4 mm deep on a female face and 6 mm deep on a male face. Effacement of the labiomental groove or deepening may indicate retrognathia on one and/or macrogenia on the other because of the relative proclivity of the lower lip in relation to the labiomental fold. Vertical chin height, as well as sagittal position, will also affect labiomental fold morphology.[8] Patients with a deep labiomental groove and retrognathia do not benefit from genioplasty and, indeed, genioplasty on these patients should be avoided. Instead, these patients are ideal candidates for mandibular advancement.

On the profile view, the condition of the submental area is checked for excess skin, fat and, again, the size of the submaxillary glands, as well as the position of the hyoid bone. Any abnormality of these anatomical structures should be corrected, otherwise the result of orthognathic surgery will not be ideal. Lip competence is also checked in this view. In a relaxed position, the lips are generally touching each other. Any significant separation of the lips is an indication of lip incompetence. This could result from an open bite, maxillary excess, or significant mandibular deficiency. Lip incompetence, generally, is a result of an abnormality in the skeleton, although in rare facial deformities, weakness of the musculature could account for lip incompetence. An additional basilar view of the face with the head tilted back, or an overhead view with the examiner standing above the head and checking the alignment of the different facial segments, can detect subtle deviations that might not be easily detectable with a head-on view.

Facial Width, the Malar Complex, and the Gonial Angle

Neoclassical cannons or rules to define the relationships between various areas of the head and face, suggest that the ideal intraocular distance is equal to nasal alar width and prolabial tissue length.[9] Midfacial width from zygoma to zygoma is divided into quarters, with each fourth being equal to bialar width. Farkas et al., however, found that sizable variations of horizontal measurements exist in the majority of cases.[9] Thus, neoclassical cannons of facial proportion, although helpful, cannot be accepted as the sole determinants of an aesthetically pleasing face. They are, however, valuable in rapid assessment of facial form and balance.

The bimalar distance is measured from the high point of one malar eminence to the other, which is approximately equal to the bitemporal ridge and the bigonial distance.[10,11] The widest point of the face, the bizygomatic arch distance, is just lateral to the zygomaticotemporal suture. The angulation of the malar-midface complex from the horizontal plane measures approximately 35–45 degrees.[10,11] Whitaker suggests evaluating the malar-midface in thirds: medial, middle, and lateral.[10,11] Each zone is observed for relative symmetry and fullness (Fig. 12.5). The malar-midface inclination is approximately 45 degrees, extending from the zygomaticotemporal suture to the nasolabial fold, if the angle is measured from the Frankfort horizontal.[10–12] The gonial angle is 105–115 degrees in the ideal face.[11] By the aesthetic standards of profile-plasty, as advocated by Gonzalez-Ulloa, the frontonasal angle, lower lip, and the chin point are in a vertical plane with one another.[6]

Highlight-Lowlight Areas

Highlight-lowlight areas that correspond to anthropologically definable soft tissue measurements based on boney structures have been identified.[10] The highlight areas are the temporal ridge—supraorbital ridge structures, the malar-midface structures, and the mandible-chin structures. The lowlight areas are those lying between, above, and below. With the application of craniofacial surgical principles, the lowlight areas can be potentially further deemphasized, while the highlight areas are increasingly highlighted. Adjuvant maneuvers such as buccal lipectomy should be considered during routine orthognathic surgical procedures.

Vertical Proportions

Clinical evaluation of vertical proportions can be conveniently divided into equal thirds (Fig. 12.3). The trichion to the supraorbital ridges represents the upper third; the supraorbital ridge to the subnasale represents the middle third; and the subnasale to the menton represents the lower third. The lower third may be further divided: subnasale to stomion, representing one third, and stomion to menton, two thirds. These proportions are derived from the classical cannons and as already mentioned, variation is the norm. The use of the golden rule as described by Ricketts is an excellent means of analyzing vertical dimensions.[13]

Intraoral Examination

The intraoral examination is commenced by assessing the upper lip relationship to the upper central incisors with the lips slightly parted. In an attractive face with normal occlusion and normal maxillary growth, 2–3 mm of the upper central incisors are visible between the lips.[5] After lip continence has been assessed, the amount of exposed maxillary central incisor is measured with the patient in repose and also smiling. As mentioned, ideal normal tooth show is approximately 2–3 mm at rest.[1,5] Up to 4–6 mm, however, may be attractive in some women if mentalis strain to achieve lip competence is not present (see Fig. 12.6).

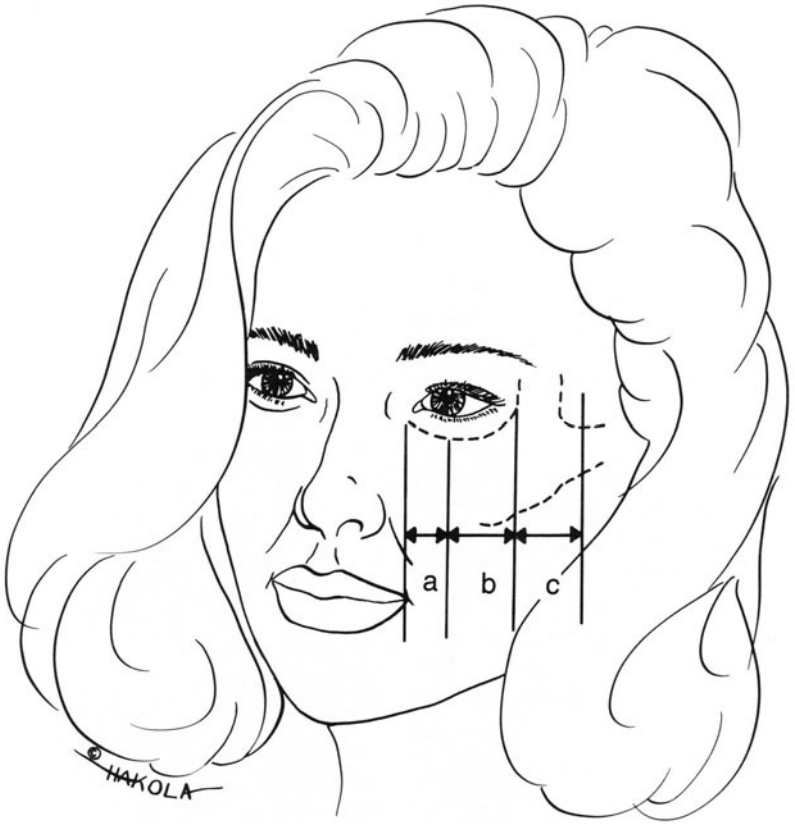

Figure 12.5. Malar zones.

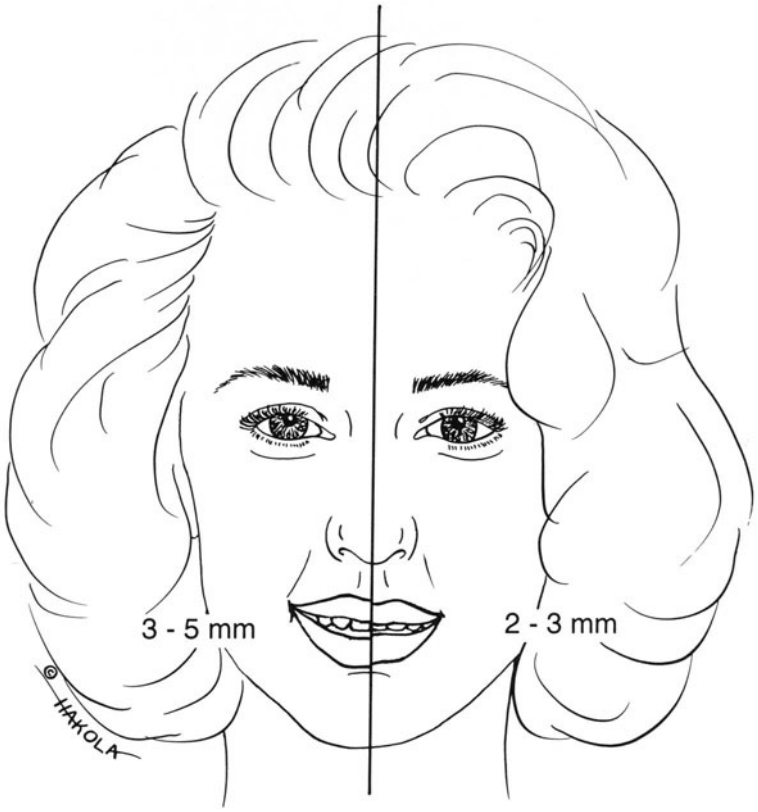

Figure 12.6. Ideal "normal" tooth show is approximately 2–3 mm at rest. Up to 4–6 mm may be attractive in some women if mentalis strain to achieve lip competence is not present.

The midline of the incisors corresponds to the midline of the face and upper lip and chin. The upper and lower incisors also have matching midlines. The patient is asked to smile, which should result in the full length of the upper incisors becoming visible with minimal or no gum show. Excessive gum show indicates an excess of maxillary growth, while insufficient incisor show reflects maxillary hypoplasia, as long as the soft tissue quantity is within normal limits. In rare occasions, the above-mentioned lip-tooth abnormalities are the result of excessive or insufficient soft tissue length of the upper lip.

Tooth alignment is then checked. In the ideal condition, the upper central incisors and bicuspids are slightly longer than the lateral incisors and the rest of the arch. The location of the palatal vault and the shape of the arch are also examined. Crowding of the teeth on the arch should be noted and, if present, may be due to abnormal dental inclinations or inadequate space in the arch. The form and size of the upper lip should also be noted. If the upper lip has a thin vermilion, maxillary repositioning may further decrease the vermilion and produce a poor aesthetic result. Treatment planning for orthognathic surgery in patients with cleft lip should take into consideration vermilion dimensions to properly plan for the amount of incisor display.

The occlusion is examined in detail and the size of the tongue is checked. Macroglossia can result in open bite, widening of the mandibular arch, crossbite, and excess mandibular growth. Conversely, an abnormally small tongue (ie., subsequent to a tongue resection) can result in collapse of the mandibular arch.

A tongue blade is then placed between the teeth and the patient is asked to bite on it. Usually, the tongue blade is positioned parallel to a line passing through the medial canthi or the pupils. If there is an asymmetric growth or rotation of the maxilla or mandible with subsequent canting of the occlusal plane, it will be revealed by this simple examination. An additional basilar view of the face with the head tilted back, or an overhead view of the examiner standing above the head and checking the alignment of the different facial segments, can detect subtle deviations that might not be easily detectable in the head-on view.

Periodontal Disease

Because the majority of patients with dentofacial deformities are adults in their 20s and 30s, special attention must be paid to the supporting structures of the teeth. Most adults in this age range will have some evidence of periodontal disease.[1] Assessment of oral hygiene and the presence of plaque, gingivitis, and periodontal pockets should be made. Orthodontic treatment is contraindicated until active periodontal disease is under control and oral hygiene is excellent. Mobility of the teeth should be checked and areas of inadequate gingival attachment noted. Referral to a periodontist may be necessary prior to further treatment.[1]

Dentition

Dental health is a prerequisite before combined orthodontic-surgical treatment. Although extensive crown, bridge, and occlusal rehabilitation should be postponed until after basilar-occlusal relationships are established, careful consideration must be given to prosthodontic needs at the time of initial planning. Missing teeth and active caries lesions should be identified and treated.

Occlusal Relationships

The occlusal relationships are evaluated after assessment of the maxillary and mandibular dental arch for crowding and spacing problems.[1,5] An occlusal slide from centric relation to centric occlusion is usually associated with a functional shift of the mandible.[1,5] The amount and direction of the mandibular shift are important findings. Horizontal incisor overjet and vertical overbite should also be noted. Angle classification at the molar and canine levels should be established and posterior and anterior crossbites recorded.

Masticatory Function

Temporomandibular joint disorders may coexist with dentofacial deformities. Clinicians should therefore thoroughly document the function of the masticatory apparatus prior to treatment.[1] Symptoms of temporomandibular joint disorders are obtained by asking the patient appropriate questions, including any history of clenching or grinding of teeth, headaches, muscle pain, limitation of opening, locking open or closed, temporomandibular joint popping, or clicking. Their duration, time of day, identifiable cause, constitutional symptoms, past treatment for conditions, and any factors that improve or worsen the symptoms must also be recorded.[1] Signs of dysfunction should be noted during clinical examination. The muscles of mastication should be individually palpated and areas of tenderness noted. The temporomandibular joints should be palpated both externally and/or intraorally at rest and during function. Clicking, crepitus, and catching should be accurately described.[1] The interincisal dimension, lateral excursions, and protrusive excursion should be measured and recorded. The pattern of mandibular opening and closure and any deviation should be assessed, especially if deviation coincides with the palpable or audible click of the temporomandibular joint.

Normal mandibular opening is measured from incisal edge to incisal edge, and the overbite (approximately 2 mm) is included. This measures 40–56 mm in the normal individual. The normal range of maximal lateral movement at 1 cm opening is 9–13 mm.[13]

Radiologic Examination

Analysis of radiographs will provide the clinician with information to confirm the clinical findings and supplement the clinical examination.[1] Screening radiographs basic to any orthognathic procedure include: (1) a lateral and posterior/anterior cephalogram and (2) a panoramic radiograph. By tracing and measuring the lateral and posterior/anterior cephalograms, one may objectively evaluate the boney and soft tissue morphology. Detailed cephalometric analysis will be discussed in Chapter 13. The panoramic radiograph provides general information on the temporomandibular joints and any abnormalities or suspected pathology. In addition, it defines the number and position of both erupted and unerupted teeth. When abnormalities are detected, specific radiographs such as periapicals for the teeth or tomograms to more clearly delineate the TMJ, can be ordered. When segmental maxillary surgery is planned, periapical radiographs are necessary to visualize the proximity of the tooth roots to the proposed lines of osteotomy.

Summary

Planning the skeletal, soft tissue, and dental correction of craniomaxillofacial deformities requires careful collection of data from numerous sources. The medical and dental history, and clinical examination, are essential first steps in the detailed treatment plan that will eventually be formulated. With this information in mind, the chapters on cephalometric analysis and orthognathic surgery will complete the basic steps toward understanding the surgical management of maxillofacial deformities.

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CHAPTER 13

Cephalometry and Cephalometric Analysis

James W. Ferraro

Cephalometry, as defined by *Dorland's Illustrated Medical Dictionary*,[1] is the scientific measurement of the dimensions of the head.

Ever since Camper investigated prognathism craniometrically in 1791, anthropologists have been interested in facial form. Anthropologists began studying the skull on dried specimens. By measuring the size of various parts and recording variations in position and shape of cranial and facial bony structures, it became possible to devise broad standards descriptive of the human head. Certain landmarks and measurement points were developed. However, the fact that these measurements were made on nonliving subjects presented a problem when trying to analyze the living patient with a skull or jaw abnormality.

Progress was made when Simon,[2] using photographs, developed gnathostatics as a diagnostic means of relating teeth and their bases to each other and to craniofacial structures. The use of photographs, however, limited the evaluation method and hence the work of Todd[3] and Broadbent.[4] These gentlemen pioneered the use of roentgenographic cephalometrics, which combined the face-conscious superficial evaluation of Simon with the anthropological measurements of the underlying bony structures of the living individual (see Fig. 13.1).

The use of cephalometrics requires a carefully oriented and anterior–posterior headplate. The patient sits in a lead holder perpendicular to the floor and parallel to the film cassette, called a cephalometer. The distance from x-ray machine to the center of the skull is 5 feet, with 11 inches between the center of the head and the film.

This x-ray technique sets the standard so that repeated x-rays are possible without the effect of magnification. Because cephalometric radiography is a standardized technique, repeated head films of the same patient may be traced on clear acetate paper and compared. These radiographs can therefore be used for immediate clinical evaluation, longitudinal growth studies, and in the postop evaluation of patients to see whether our goals were attained and whether long-term stability has been achieved.

The use of cephalometric radiography is only one of the tools we use to evaluate the patient for orthognathic surgery. Even though x-rays may be helpful in evaluating the skeleton, dental relationship, and soft tissue structures, the physical examination of the patient's soft tissues and the use of dental stone models are also imperative because these help us in our three-dimensional evaluation of the patient (see Fig. 13.2).

Classification of Malocclusion

Malocclusion can be divided into three groups: (1) dental dysplasias, (2) skeleto-dental dysplasias, and (3) skeletal dysplasias.

Dental dysplasias are malocclusions that exist strictly because the teeth are abnormally related to each other. Here the jaw relationship (skeleton) is normal with good muscle balance, but the arches lack the space to accommodate the teeth. Incisors may be rotated, crowding may prevent some of the teeth from erupting into the arch, and other teeth may be bucally or lingually displaced.

In skeleto-dental dysplasias, the malocclusion involves teeth that are in malposition, and also the skeletal relationships are abnormal. The maxilla and mandible are in an abnormal position with each other or to the cranial base. An example would be a crowded

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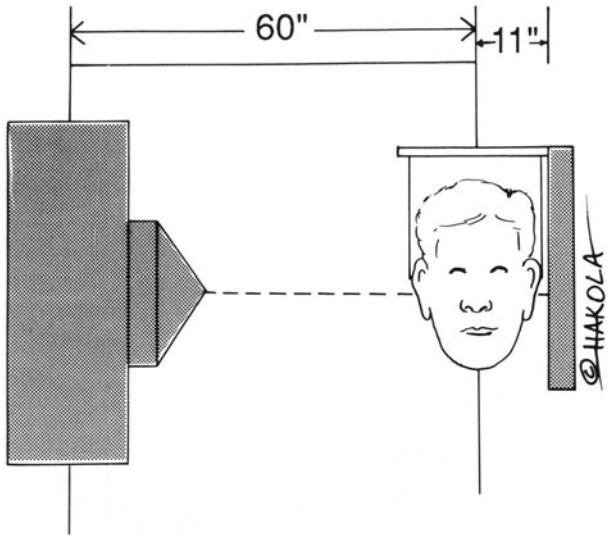

Figure 13.1. Schematic of standard radiograph cephalometer design.

linear arch that is also short in its anterior-posterior dimensions.

In skeletal dysplasias the teeth are in good arch alignment, but the maxilla and/or mandible may be small or long in its superior inferior dimension.

The major thrust of the classification of malocclusion was introduced by Edward H. Angle in his classic article in *Dental Cosmos* in 1899.[5] The basis for the Angle classification was his hypothesis that the first molar is the key to occlusion.

He wrote

> All teeth are essential, yet in function and influence some are of greater importance than others, the most important of all being the first permanent molars . . . They are by far the most constant in taking their normal positions . . . especially the upper first molars . . . which we call the keys to occlusion. We believe that nature so rarely errs in the location of the upper first molars—the very cornerstone as it were in the foundation of the structure of an organ so essential to the whole physical economy as a dental apparatus—as to make it a matter of little or no concern to us except, possibly in research work. (p. 248)

From this work has evolved Angle's classification of malocclusion, which we frequently refer to today when classifying abnormal dental and skeletal relationships. One important consideration to keep in mind is that when Angle wrote his classification it was in reference to malocclusions in children.

The Class I neutroocclusion that we consider the ideal refers to a normal molar relationship, but in his classification the anterior teeth were abnormally positioned. From here on in this chapter the reference to Class I, however, refers to a normal molar and cuspid relationship with no dental discrepancies.

Class I (Neutroocclusion) (Fig. 13.3)

1. The mesial buccal cusp of the upper first molar articulates in the buccal groove of the lower first molar.

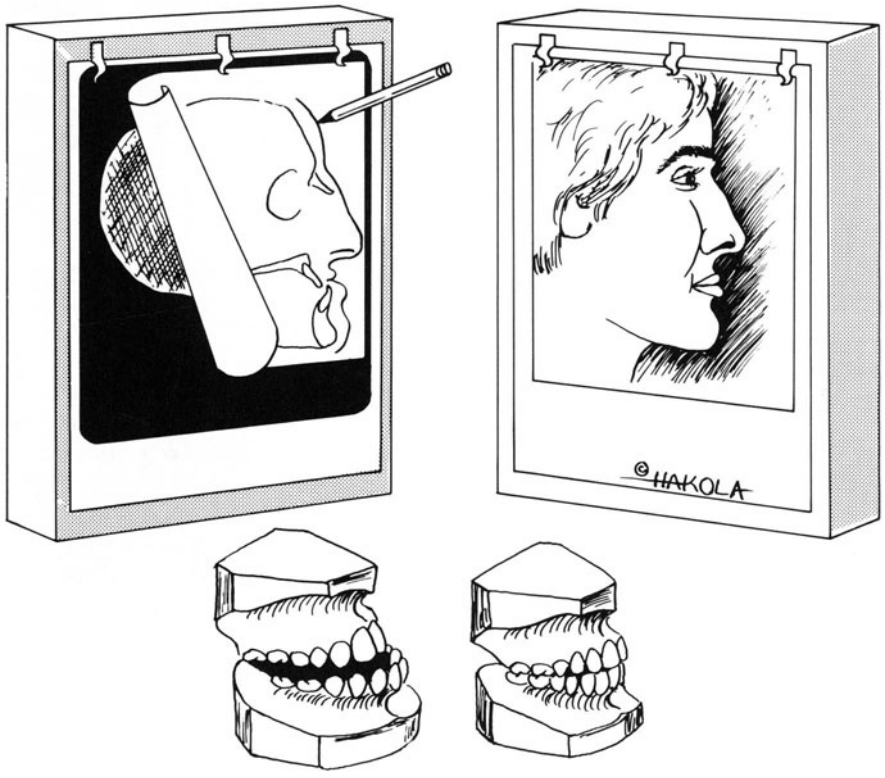

Figure 13.2. Parameters used to evaluate the patient. It takes into account the skeleton, soft tissue drape, and dental relationships.

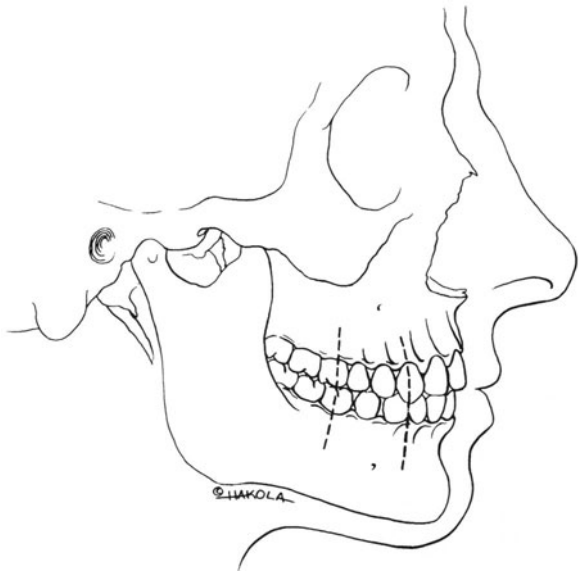

Figure 13.3. Class I Neutroocclusion. Malocclusion confined to the anterior teeth.

2. The maxillary cuspid interdigitates between the mandibular first bicuspid and cuspid.
3. May be associated with bimaxillary protrusion and, in fact, most cases of bimaxillary protrusion are associated with a Class I molar and cuspid relationship.

Class II (Distoocclusion) (Fig. 13.4)

1. The mesial buccal cusp of the maxillary first molar articulates between the lower first molar and the second bicuspid. Hence the term distoocclusion, because the mandible is distally placed or retropositioned when compared to the maxilla.

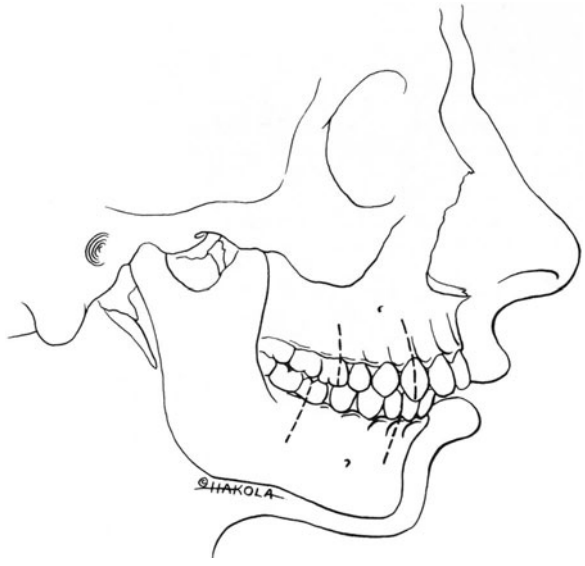

Figure 13.4. Class II Distoocclusion. Malocclusion may be unilateral with either pro- or retrusive central incisors.

2. The mandibular cuspid is distal to the maxillary cuspid and articulates between the maxillary first bicuspid and cuspid.
3. There are two subdivisions of Class II malocclusions.
   A. Class II, Division 1—Here there is a Class II molar relationship but it is also associated with:
      a. The maxillary arch which is usually u-shaped, but here is v-shaped with narrowing of the bicuspid and cuspid areas.
      b. Flaring or labioversion of the maxillary incisors.
      c. There is an increased overjet and therefore the lower lip is rolled out under the maxillary incisors.
      d. There is frequently poor lip seal and therefore an associated mentalis strain in an attempt to get lip closure.
      e. Excessive curve of Spee, which results in a deep bite.
      f. Eversion of the vermilion of the lower lip with an increase in the labiomental crease.
   B. Class II, Division 2—Here also there is a Class II molar relationship but it is associated with:
      a. There is excessive eruption of the mandibular incisors (supraversion).
      b. The maxilla is usually wider than normal especially in the canine area.
      c. There is excessive labial inclination of the maxillary central incisors, with excessive lingual inclination of the maxillary lateral incisors.
      d. Sometimes the four anterior maxillary teeth have a lingual inclination and the cuspids have a labial inclination.
      e. Usually a deep bite with the teeth of the mandible frequently striking the palatal mucosa.
      f. These lingually displaced anterior teeth in the maxilla frequently result in a "locked" type of occlusion, with the condyle being forced posteriorly in the glenoid fossa with each mandibular closure.

Class III (Mesioocclusion) (Fig. 13.5)

1. Here the mesial buccal cusp of the maxillary first molar articulates between the first and second mandibular molar teeth. Hence the term mesioocclusion, because the mandible is mesially placed or prognathic when compared to the maxilla.
2. The mandibular cuspid articulates between the maxillary lateral and cuspid teeth.
3. The lower incisors usually have significant dental compensations and are lingually inclined.
4. There is an end-to-end or cross-bite relationship in the anterior teeth.
5. The maxillary arch is frequently constricted and, as a result, you may see a total arch cross-bite or, in more

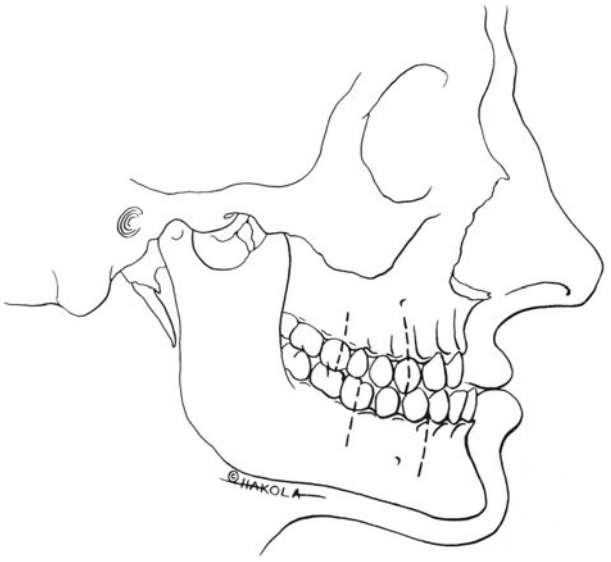

Figure 13.5. Class III Mesioocclusion. Malocclusion may include maxillary insufficiency, mandibular overgrowth, or a combination of both.

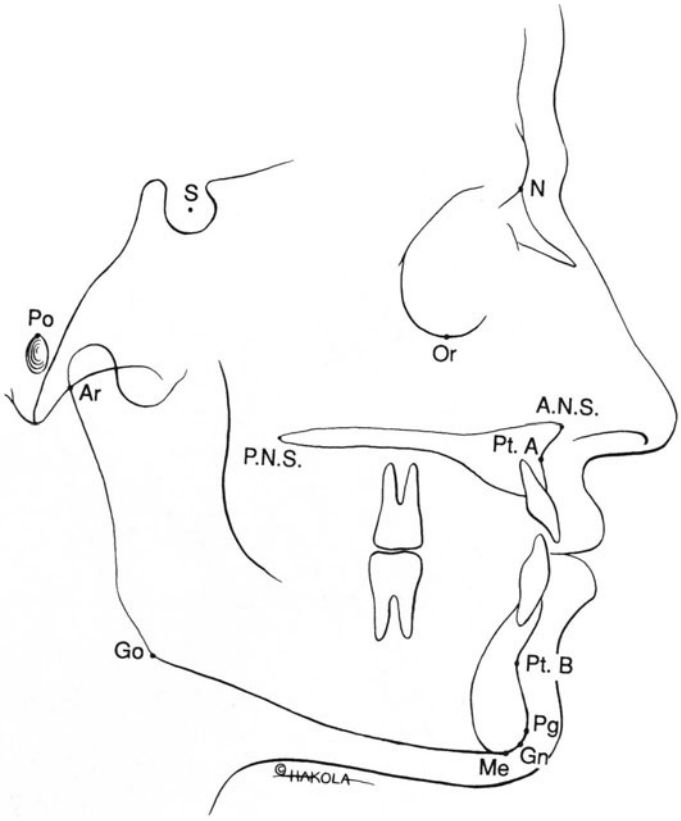

Figure 13.6. Cephalometric boney landmarks.

severe cases, a maxillary arch totally within the maxillary dentition.

6. The lower lip is full, especially just below the vermilion border, and the vermilion is rolled inward.
7. The upper lip is curled up under the lower lip and, as a result, less vermilion is evident on frontal view.

Skeletal Assessment

Using the boney landmarks as seen in Fig. 13.6 and Table 13.1 lines are drawn to create planes (see Figs. 13.7 and 13.8). These planes are now joined to form angles. The use of angles is to decrease the effect of magnification and therefore make the measurements more accurate and, as a result, more reproducible.

Mandibular Length–Ar-Pg (Fig. 13.8)

The mandibular length of 115 ± 5mm is a useful distance measurement to evaluate the length of the mandible, especially when true versus pseudoprognathism is being considered.

The Facial Plane Angle—Frankfort Horizontal (Po-Or) and the Facial Plane (Fig. 13.9)

The mean for this angle is 88 ± 3 degrees. This angle is influenced by the chin position. If there is a microgenia or a retrogenia, this angle will tend to be more acute. As the chin becomes more prominent it becomes more obtuse. This angle, however, can also be affected if there

Table 13.1. Cephalometric landmarks (boney) (Fig. 13.6).

S	(*Sella*) the center of the pituitary fossa—Sella Turcica
N or Na	(Nasion)—the most anterior point at the junction of the nasal and frontal bones in the mid saggital plane.
Po	(*Porion*) the point located at the most superior point of the external auditory meatus.
O or Or	(*Orbitale*) the lowest point on the inferior boney border of the left orbital cavity as viewed from the lateral aspect.
ANS	(*Anterior Nasal Spine*) most anterior tip of maxillary nasal spine.
PNS	(*Posterior Nasal Spine*) midline tip of posterior spine of hard palate in the mid saggital plane.
P or Pog	(*Pogonion*) the most anterior point on the contour of the mandibular symphysis.
Pt. A	(*Point A*) the deepest midpoint on the maxillary alveolar process between ANS and the crest of alveolar ridge.
Pt. B	(*Point B*) the deepest midpoint on the alveolar process between the crest of the ridge and pogonion.
Me	(*Menton*) the lowest point on the contour of the mandibular symphysis.
Gn	(*Gnathion*) the most anterior–inferior point on the chin contour constructed point, determined by bisecting the angle formed by the facial and mandibular planes.
Ar	(*Articulare*) the junction of the basisphenoid with the posterior of the condyle of the mandible.
Go	(*Gonion*) the point at the angle of the mandible that is most inferiorly and posteriorly directed.

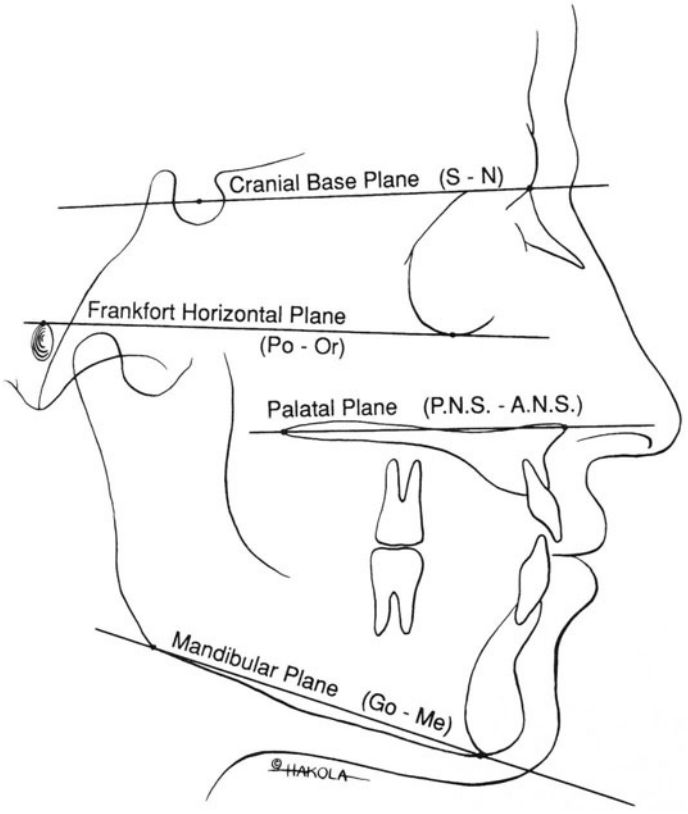

FIGURE 13.7. Horizontal planes.

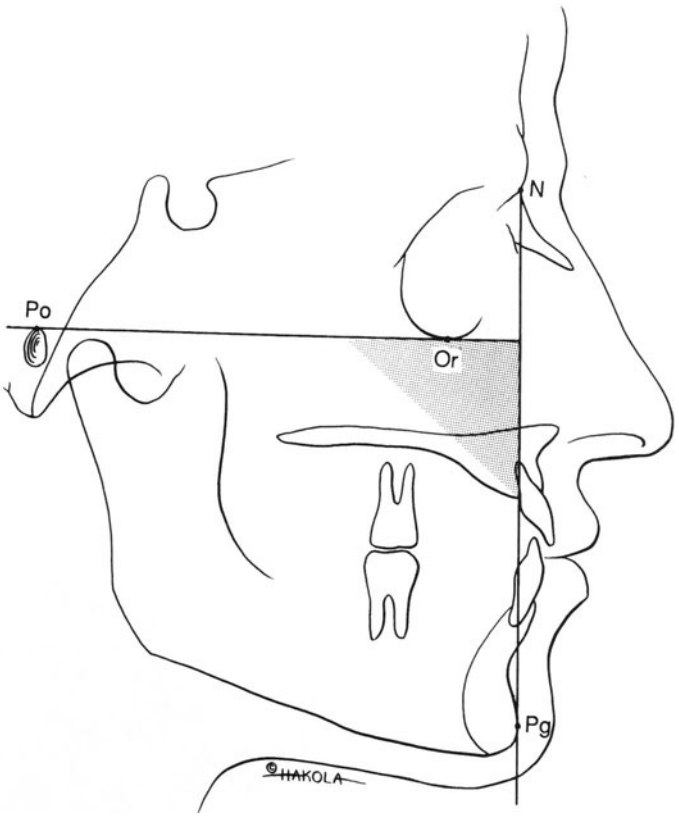

FIGURE 13.9. Facial plane angle. Frankfort Horizontal–Facial Plane (mean 88° ± 3°).

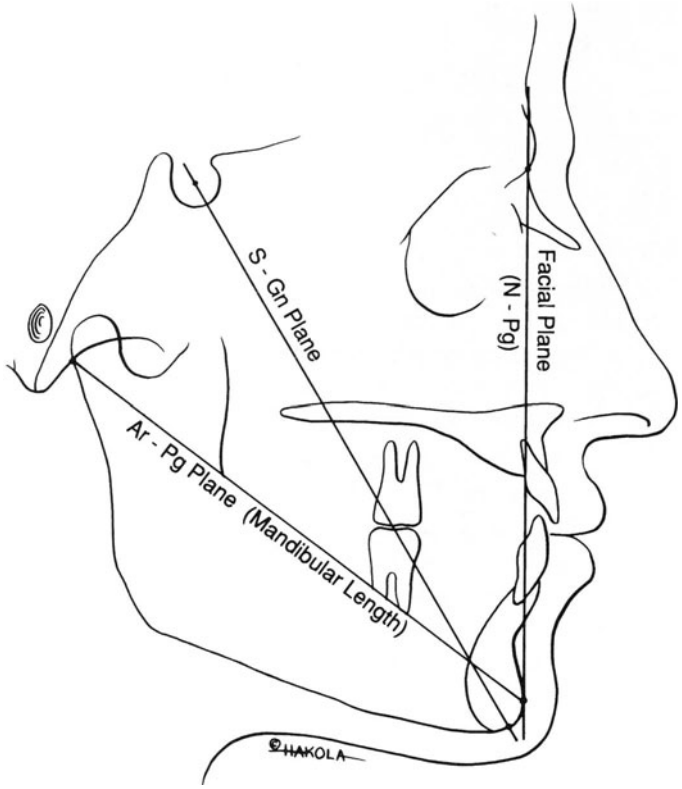

FIGURE 13.8. Vertical planes.

is retrognathia, as in Class II, or prognathia, as in Class III, both of which would affect the amount of chin projection.

S–N–A Angle (Fig. 13.10)

The mean for this angle is 82 ± 3 degrees and it relates the maxilla with the cranial base. In cases of maxillary retrusion this angle would be more acute. However, in some craniofacial deformities the cranial base may be too steep. Therefore there is a check angle that we use to better evaluate the maxilla: the Landes angle or the angle of maxillary depth.

Landes Angle (Angle of Maxillary Depth) (Fig. 13.11)

This angle is formed by the intersection of the Frankfort horizontal and the N–A plane. The mean is 88 ± 3 degrees. The angle is a check on the S–N–A, but it is more reliable because it relates to the reliable Frankfort Horizontal Plane. The angle also evaluates the anterior-posterior position of the maxilla and helps to determine whether a Class II or Class III malocclusion is secondary to a malpositioned maxilla or mandible.

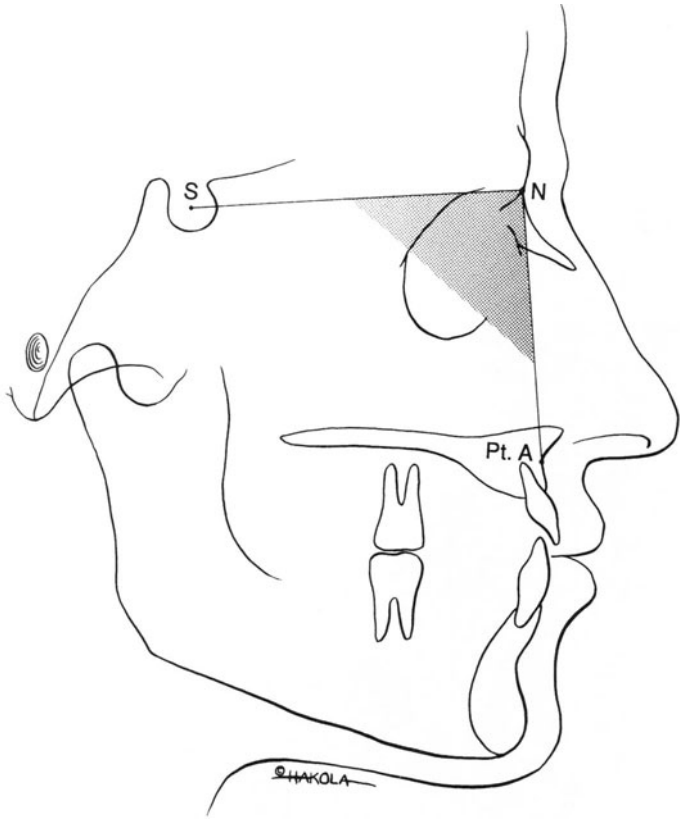

Figure 13.10. S–N–A angle (mean 82° ± 3°).

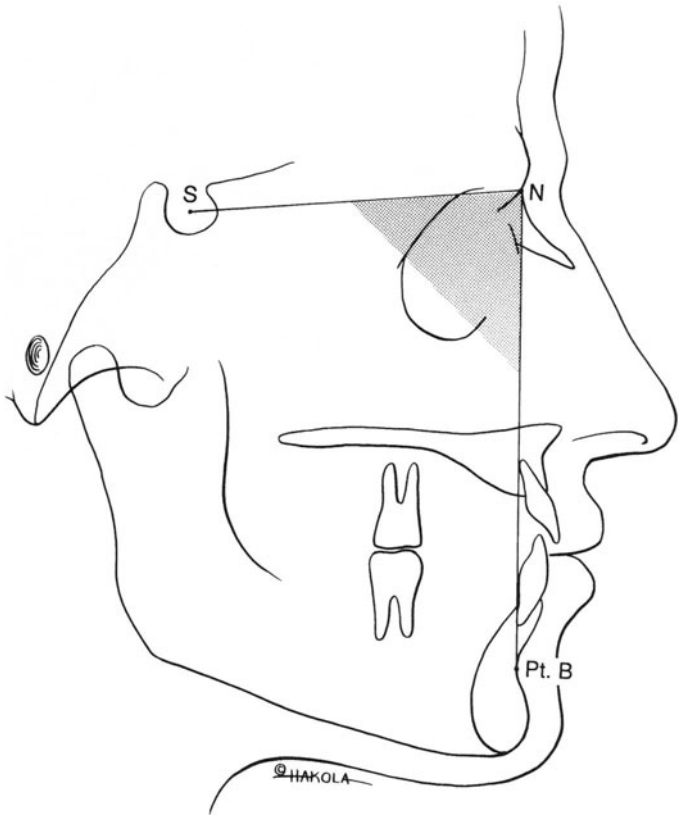

Figure 13.12. S–N–B angle (mean 80° ± 3°).

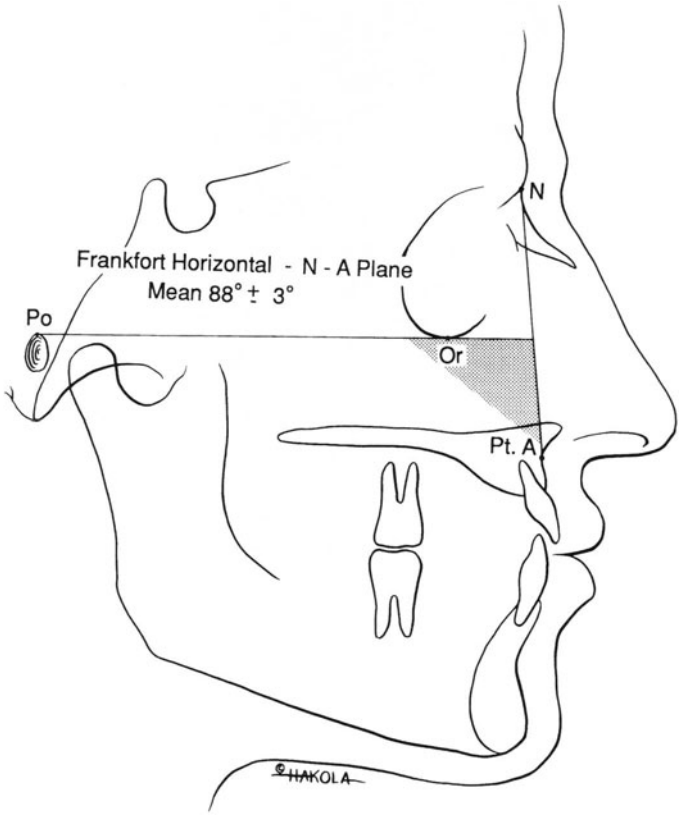

Figure 13.11. Landes angle (angle of maxillary depth).

S–N–B (Fig. 13.12)

This angle has a mean of 80 ± 3 degrees. It relates the mandible to the cranial base. It is directly affected in Class III malocclusions by being more obtuse. In cases of retrognathia associated with Class II malocclusions it will become more acute.

Angle of Convexity (Fig. 13.13)

The angle of convexity is the superior angle formed by the N–A plane and the Pg-A plane. The mean is 0 ± 8 degrees. It determines the convexity of the patient's face and is influenced by a retruded or protruded maxilla and the chin position. It Pt. A is anterior to Pt. B, then the angle is positive. However, if Pt. A is posterior to Pt. B then the angle would be a negative number. In Class II malocclusions or pseudoprognathic jaw deformities the angle of convexity would usually be negative. In cases of mandibular retrusion the angle would be positive.

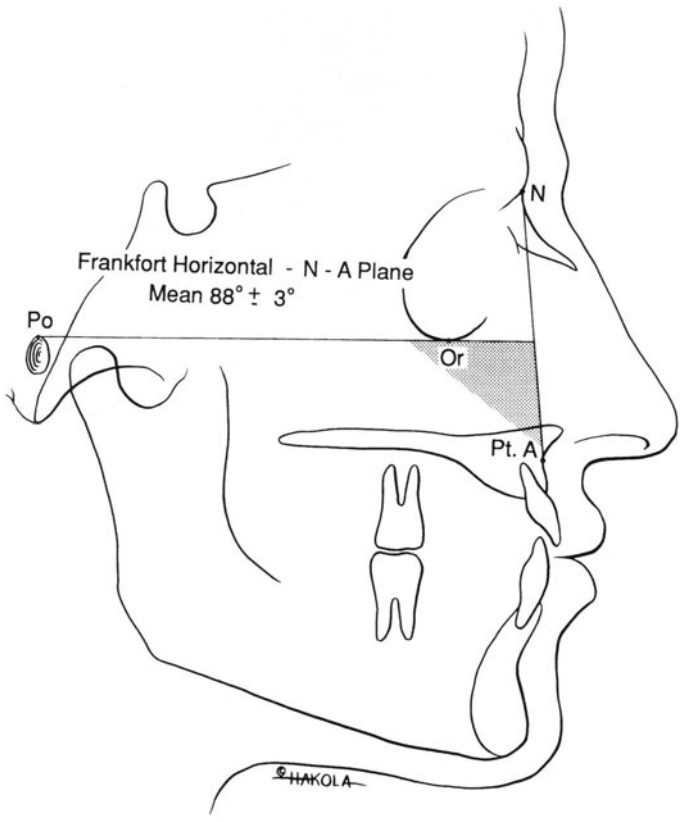

FIGURE 13.13. Angle of convexity (mean of 0° ± 8°).

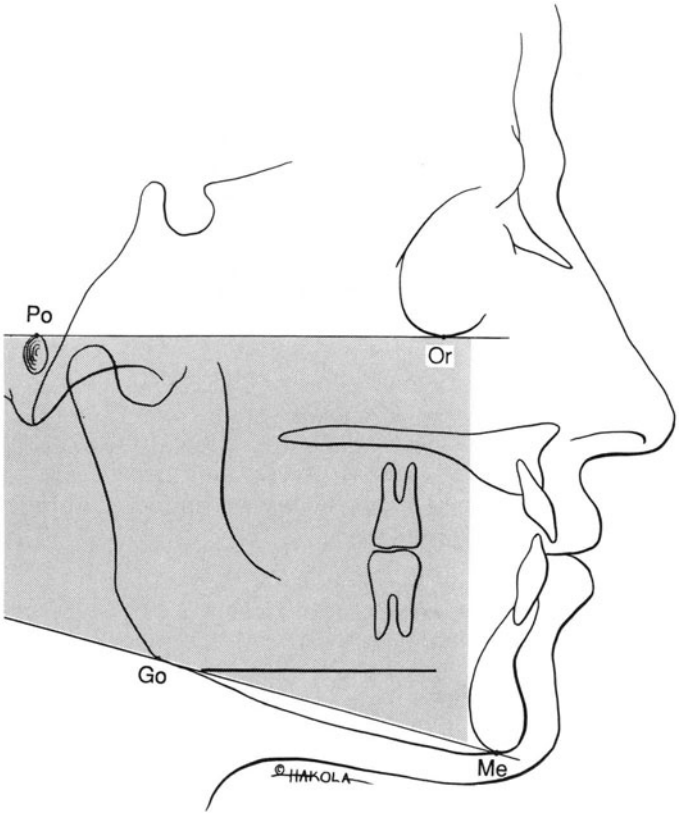

FIGURE 13.14. Mandibular plane angle (FMA) (Frankfort horizontal–mandibular plane, mean 21° ± 3°).

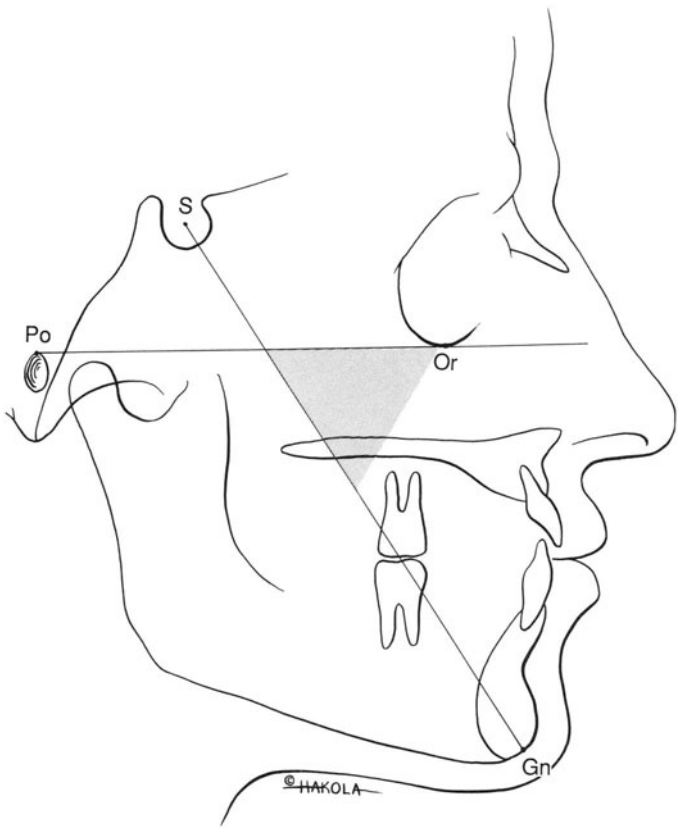

FIGURE 13.15. Y-axis (growth axis). S-Gn (Y-axis)–Frankfort horizontal (mean 59° ± 6°).

Mandibular Plane Angle (Frankfort Horizontal-Mandibular Plane (Fig. 13.14)

This angle is formed by the intersection of the Frankfort Horizontal and the mandibular plane. The mean is 21 ± 3 degrees. It is an angle that expresses the vertical posterior facial height in relation to the anterior facial height. In vertical discrepancies such as open bite deformities, it is obtuse, whereas in short face syndrome patients, the angle is more acute. According to Epker and Fish,[7] patients who have acute angles tend to have strong musculature and deep bites, whereas patients who have high or obtuse angles have weak musculature.

Y-Axis (Growth Angle) (Fig. 13.15)

This is the anterior–inferior angle formed by the S-Gn plane and Frankfort horizontal. It is a predictor angle used by orthodontists to predict the amount of forward and/or downward growth of the mandible. If the angle is obtuse it indicates the mandible is tending to grow downward rather than forward. Interceptive orthodontics would attempt to correct this problem early. If the angle is acute it indicates that the mandible is growing forward, resulting in a prognathic tendency or a vertically deficient face.

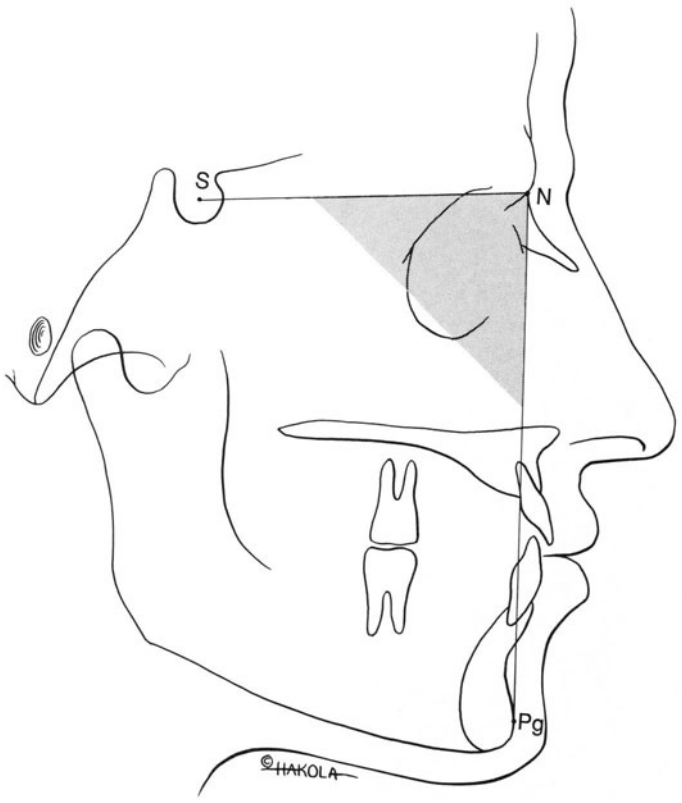

FIGURE 13.16. S–N–Pg (mean 80° ± 3°).

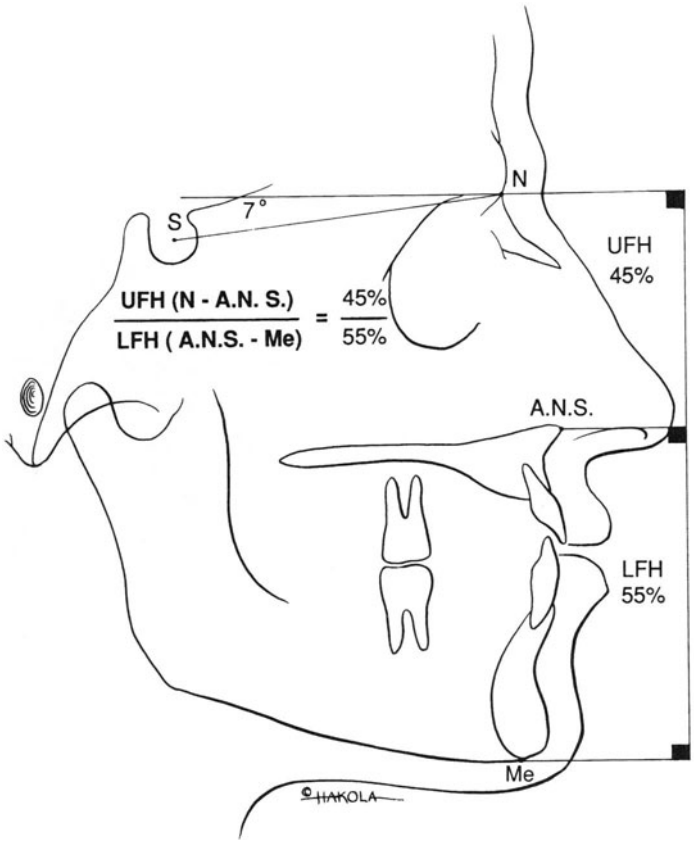

FIGURE 13.18. Upper and lower facial height using A.N.S.

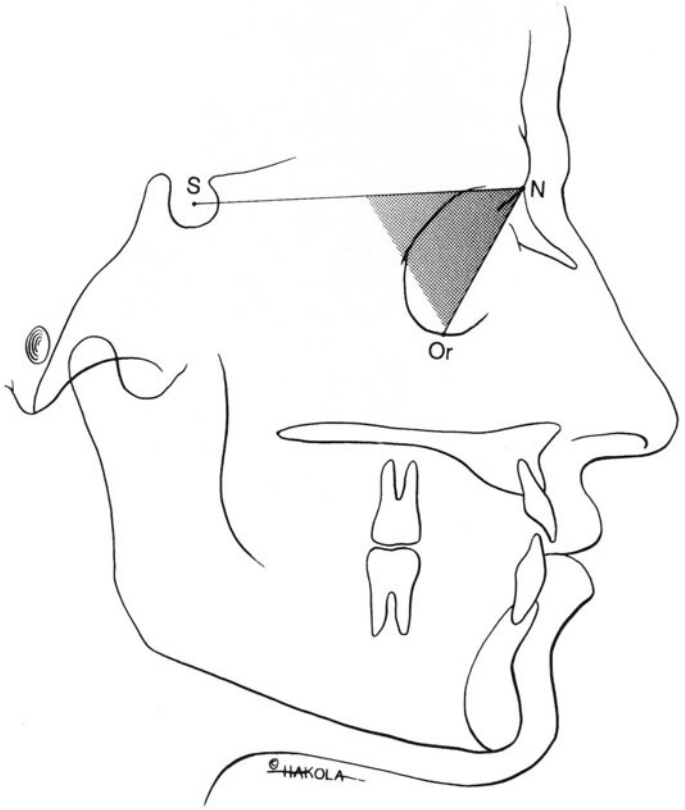

FIGURE 13.17. S–N–Or (mean 54° ± 4°).

S–N–Pg (Fig. 13.16)

This angle relates the cranial base plane with the facial plane. The mean is 80 ± 3 degrees. It is a less accurate measurement than the facial plane angle, but can be used as a check measurement to see the accuracy of the facial plane angle. It relates primarily the chin with the cranial base but is also affected by the position of the mandible as a whole.

S–N–Or (Fig. 13.17)

This angle relates the cranial base with the orbital rim position. The mean is 54 ± 4 degrees. This angle is helpful if one is concerned about the orbital rim position in relation to maxillary hypoplasia.

Upper Facial Height, Lower Facial Height, and Total Facial Height (Figs. 13.18 and 13.19)

Measurement of the total facial height is evaluated by first drawing a plane 7 degrees from the S–N plane. This is known as the horizontal plane and comes from the work of Burstone[8] in 1958, later modified by Legan and Burstone[6] in their paper in 1980. From this horizontal plane (HP) a perpendicular line is dropped and horizontals are made to either ANS or Pt. A and menton.

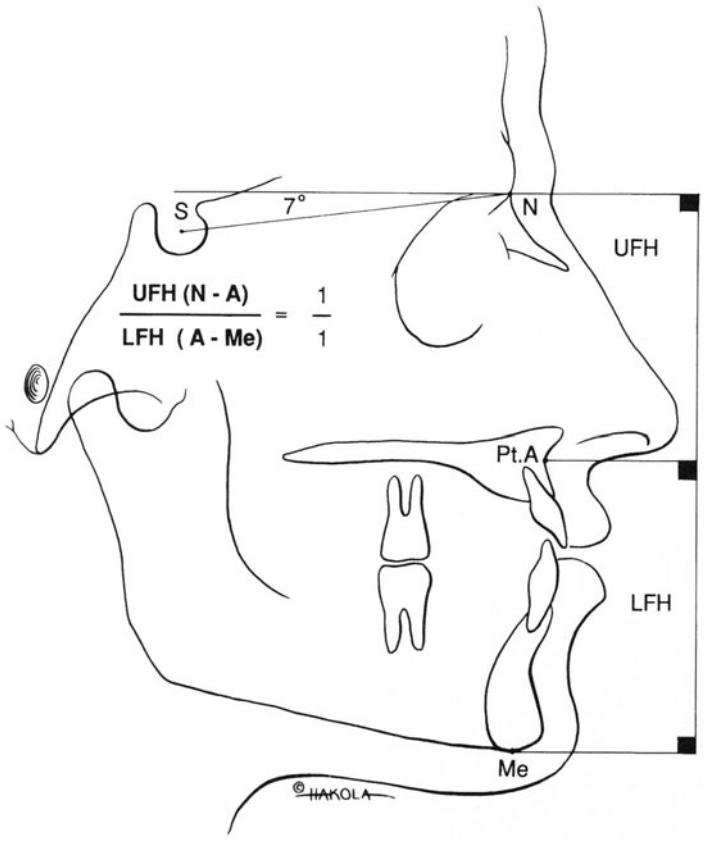

Figure 13.19. Upper and lower facial height using Pt. A.

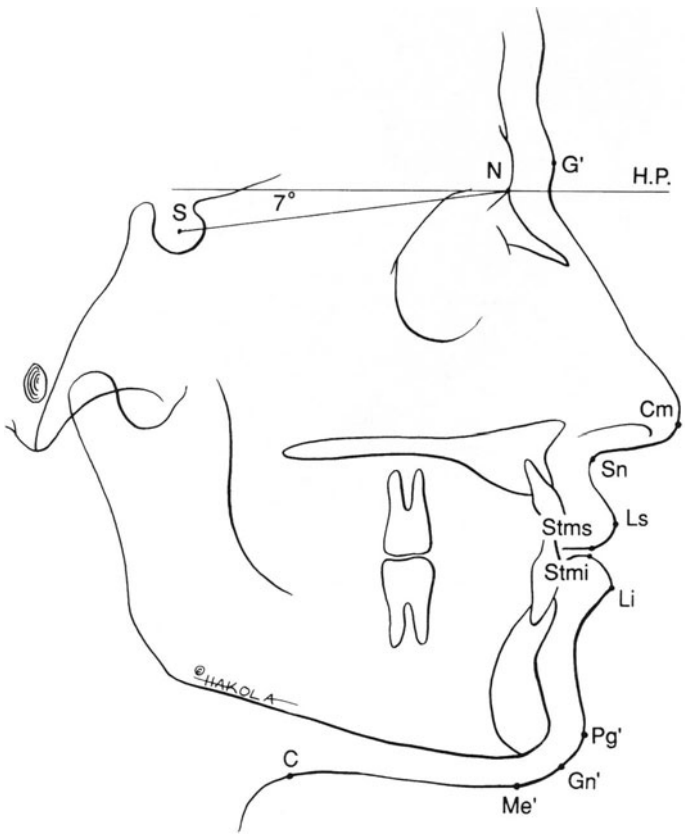

Figure 13.20. Soft tissue landmarks.

If you are using ANS related to nasion, then this distance represents 45 percent of the total facial height, whereas if you are using Pt. A to nasion, it represents about 50 percent of the total facial height. In an article by Grayson,[9] means and extremes in total and lower facial height in linear measure are evaluated as follows:

	Male	Female
N-Me (mm)	137 ± 8 mm	112 ± 5 mm
ANS-Me (mm)	79 ± 6 mm	69 ± 5 mm

Soft Tissue Analysis

In evaluating the soft tissues of a patient we again turn to the work of Legan ad Burstone.[6] It has been found that in planning surgery, changes in soft tissue are important to the final outcome of the procedure. It is also important to the final outcome of the procedure. It is also important to determine whether the procedures will compromise the soft tissues of the neck, nose, or lips so as to produce a result that may be dentally correct but cosmetically a disaster. Figure 13.20 shows the soft tissue points and Table 13.2 defines each point.

Angle of Soft Tissue Facial Convexity—G'-Sn-Pg' (Fig. 13.21)

This is the inferior angle formed by the soft tissue glabella (G') and subnasale plane with the subnasale soft tissue pogonion (Pg') plane. The mean is 12 ± 4 degrees.

Table 13.2. Cephalometric landmarks (soft tissue) (see Fig. 13.20).

G'	(*Soft Tissue Glabella*) the most prominent point in the midsagittal plane of the forehead.
Cm	(*Columella point*) the most anterior point on the columella of the nose.
Sn	(*Subnasale*) the point at which the nasal septum merges with the upper cutaneous lip and the midsagittal plane.
Ls	(*Labrale superius*) the mucocutaneous border of the upper lip in the midsagittal plane.
Li	(*Labrale inferius*) the mucocutaneous border of the lower lip in the midsagittal plane.
Pg'	(*Soft-tissue pogonion*) the most anterior point of the soft-tissue chin.
HP	(*Horizontal plane*) a plane drawn 7 degrees above the S-N plane, from which perpendicular lines are drawn to measure vertical soft tissue distances.
Stms	(*Stomion Superius*) the lowermost point on the vermilion of the upper lip.
Stmi	(*Stomion Inferius*) the uppermost point of the vermilion of the lower lip.
C	(*Cervical Point*) the innermost point between the submental area and where the neck begins its vertical position.
Me'	(*Soft Tissue Menton*) lowest point on the contour of the soft tissue chin.
Gn'	(*Soft Tissue Gnathion*) the constructed midpoint between soft tissue pogonion and soft tissue menton; can be located at intersection of subnasale to soft tissue pogonion line and the line from C to Me'.

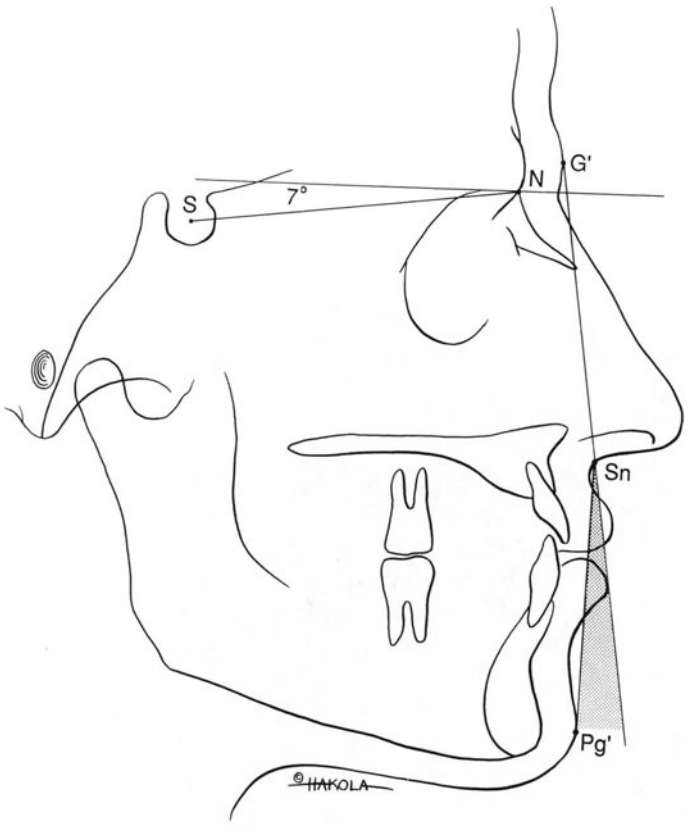

FIGURE 13.21. Angle of soft tissue convexity (G'-Sn-Pg') (mean 12° ± 4°).

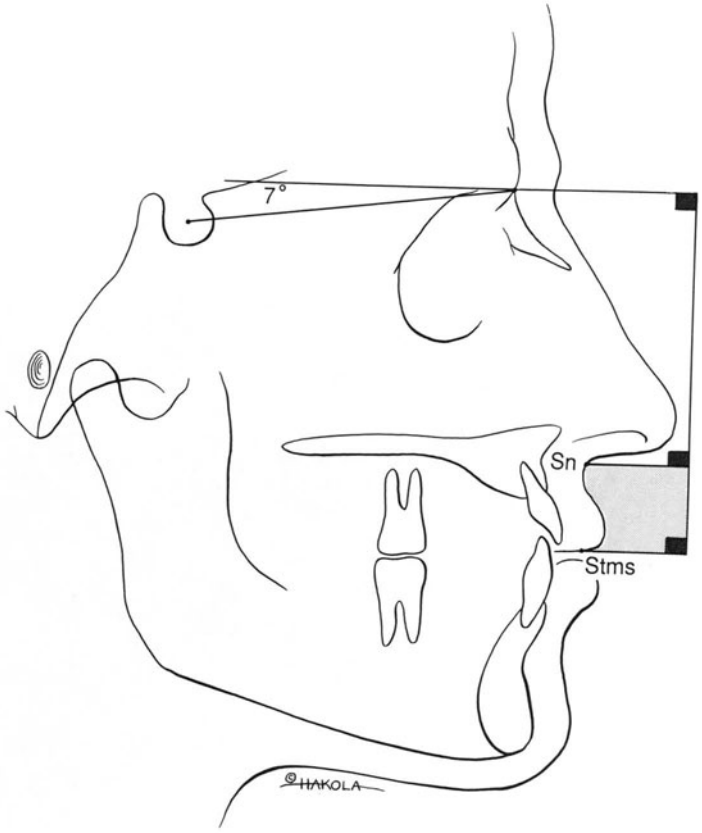

FIGURE 13.22. Upper lip length (Sn-Stms) (mean 21 mm ± 2 mm).

This angle increases as the face becomes more convex, as in patients with Class II malocclusions, but in whom the soft tissue chin button has not compensated.

Upper Lip Length (Sn-Stms) (Fig. 13.22)

This measurement, in millimeters, is the distance from subnasale to stomion superius (or the most inferior portion of the upper lip vermilion). The mean is 21 ± 2 mm. In patients with a true short upper lip this distance is below the mean and the external millimeter measurement.

Clinical Evaluation of the Soft Tissue of the Lower Half of the Face (Fig. 13.23)

a. Upper lip relation to Lower third of face $\frac{\text{Sn-Stms} = 1}{\text{Stms-me'}\ 2}$
This vertical distance measures the soft tissue of the upper lip, interdental distance at rest, and the soft tissue lower lip and chin. If the value approaches 1:3 or 1:4 it indicates a short upper lip or a long lower one third of the face. If the ratio approaches 1:1 it usually means a short lower one third of the face, and only rarely do we find a true long upper lip.

b. $\frac{\text{Subnasale-Lower Lip Vermilion}}{\text{Lower Lip Vermilion-Soft Tissue Menton}}$ $\frac{\text{Sn-Li} = 1}{\text{Li-Me'}\ 0.9}$
(Fig. 13.24). This soft tissue vertical measurement takes into consideration the interlabial distance, which at rest is 0 to 3 mm. (If this distance is wider

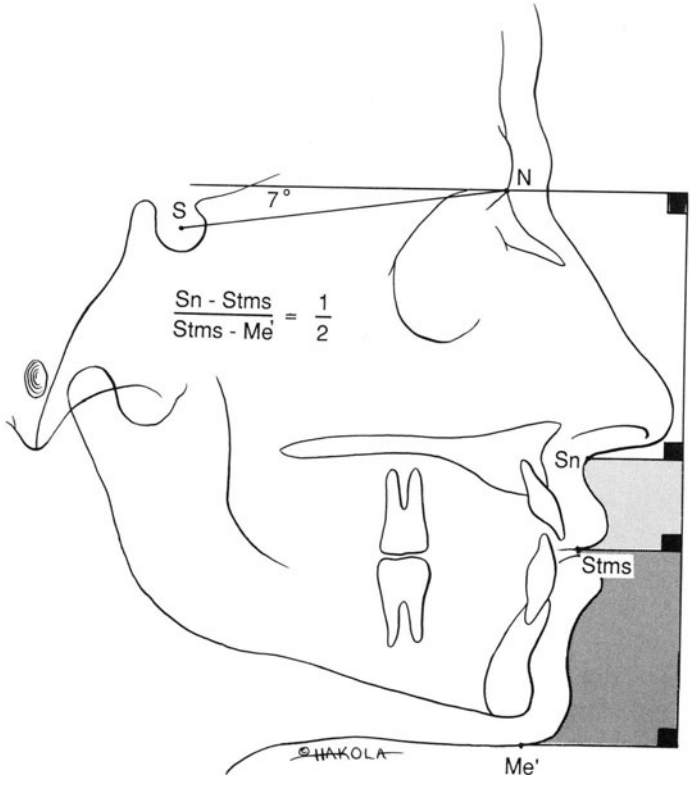

FIGURE 13.23. Upper lip relationship to lower third of face.

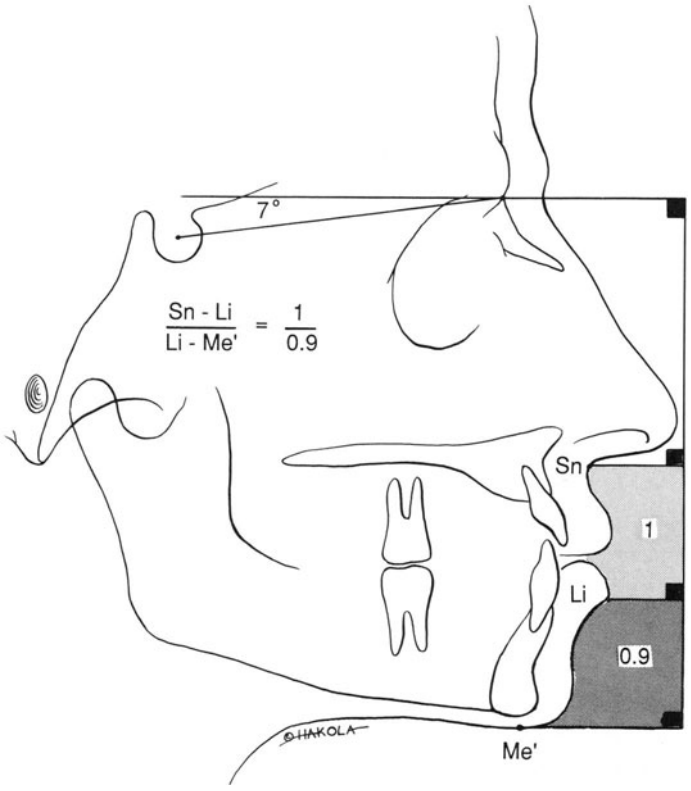

FIGURE 13.24. $\frac{\text{Subnasale—Lower Lip Vermillion}}{\text{Lower Lip Vermillion—Soft Tissue Menton}}$.

than 3 mm it indicates lip incompetence.) If this ratio is greater than 1:09, such as 3:1, it is indicative of excessive vertical dimension of the maxilla. If the ratio is smaller, such as 1:3, it is indicative of either a short maxilla in its vertical dimension or a long vertical chin.

c. $\frac{\text{Lower Facial Height}}{\text{Lower Facial Depth}} \quad \frac{\text{Sn-Gn'} = 1}{\text{Gn'-C} \quad 1}$

and Sn-Gn'-C = 100 + 7 degrees (Fig. 13.25).
The lower facial height and the lower facial depth relationship is a 1:1 relationship, and the angle formed by the lower facial throat angle is 100 ± 7 degrees. If this ratio becomes larger than 1, the patient has a short neck or if the angle is significantly greater than 100 degrees the submental area is obtuse. These two measurements become important when considering a reduction genioplasty or a mandibular setback procedure. If the setback is done in a patient with an obtuse lower facial throat angle, it could create an unsightly bulging in the submental area and a very unhappy patient. Class III patients who have short, heavy throats and obtuse lower facial throat angles should have maxillary advancement or mandibular setback procedures combined with advancement genioplasty.

d. Depth of submental sulcus (Fig. 13.26). The depth of the submental crease should be about 4 mm to produce a pleasing lower lip to chin contour.

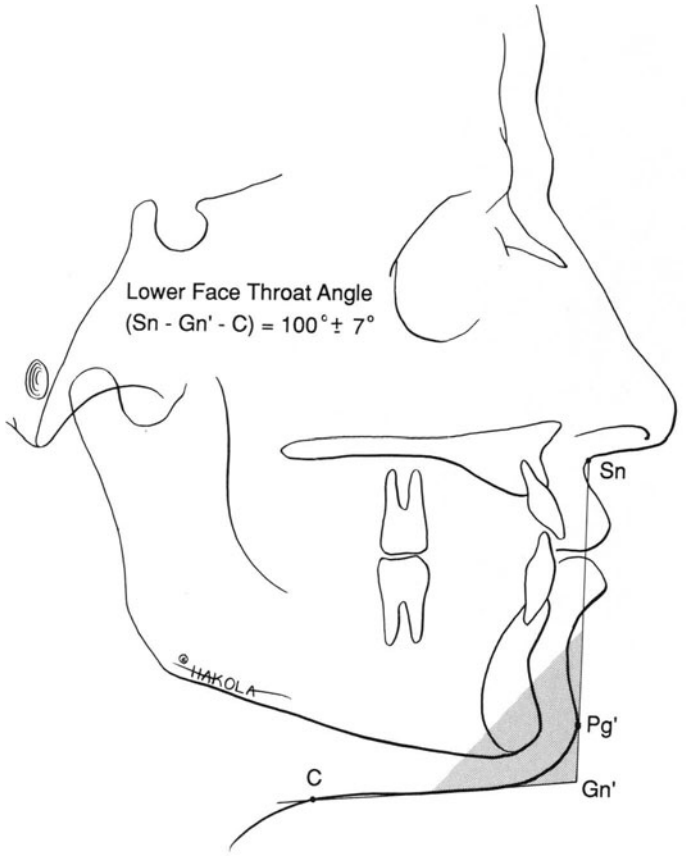

FIGURE 13.25. $\frac{\text{Lower Facial Height}}{\text{Lower Facial Depth}} \quad \frac{\text{Sn-Gn'}}{\text{Gn' Gn-C}} = \frac{1}{1}$

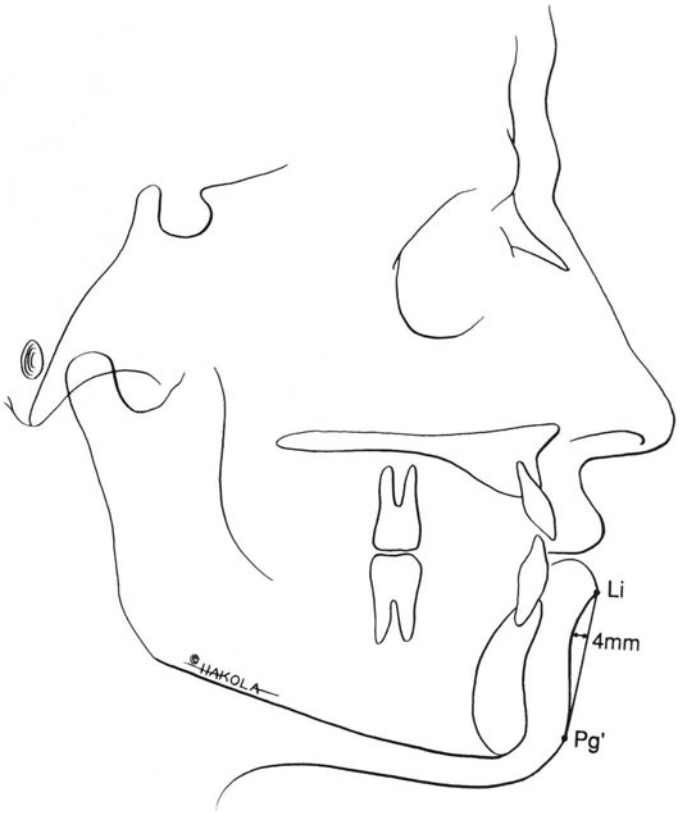

FIGURE 13.26. Labio-mental sulcus depth (depth of sulcus perpendicular to Li – Pg' = 4 mm.

Summary

With the tools of the cephalometric analysis at hand, it is possible to effectively evaluate the patient and his soft tissue drape. We can tell which jaw is abnormal and direct our surgical approach to that area. If we were to attempt to correct a pseudoprognathic jaw deformity (Fig. 13.27B) by incorrectly moving the mandible posteriorly we would doom the patient to a good functional occlusion but an aesthetically disastrous result with a flat facial appearance. As we move the various bony parts we are directly or indirectly affecting the overlying soft tissues to produce the optimum functional and aesthetic result.

1. Soft Tissue Changes in the Mandible with Advancement and Retrusion[10]
   A. A mandibular advancement of 10 mm would result in:
      a. Labial sulcus at Pt. B, moves forward 10 mm
      b. Soft tissue pogonion moves forward 10 mm
      c. The lower lip and vermilion border advance 6.5 to 8 mm. If, however, the preoperative overjet is not extensive so that the lower lip is not rolled up under the upper teeth, the lower lip advancement will be closer to 10 mm.
      d. As the mandible is advanced the labial sulcus become more shallow, and the lower lip becomes less prominent. If, however, you are doing only an advancement osseous genioplasty, the labial sulcus may remain as deep or become even more prominent.
      e. Little, if any, changes in the upper lip occur when the mandible is moved unless there is an opening of the vertical dimension. In these cases, as the increase in vertical height occurs, the upper lip becomes thinner.
2. A mandibular setback of 10 mm would result in:
   a. Soft tissue pogonion moves back 10 mm.
   b. The labial sulcus at Pt. B moves back 8 mm.
   c. Lower lip and vermilion move back 6–7 mm because they are held forward slightly by the contact with the upper incisors.
   d. Upper lip and vermilion move back 2 mm due to the increase in vertical dimension frequently seen as the mandible is moved posteriorly. (This is because prognathic patients are frequently overclosed before surgery.)
   e. The upper lip at Pt. A moves back 0.2 mm because of the change in vertical dimension. Therefore the upper lip appears to lengthen and becomes thinner.
   f. The labial mental sulcus increases slightly and the lip becomes slightly more prominent when compared to the chin and lower lip sulcus.

B. Soft tissue changes in maxilla with advancement and impaction.
   1. Maxillary advancement of 10 mm
      a. Nasal tip will advance 2–3 mm.
      b. The nasolabial angle will decrease depending on what is done or not done to the anterior nasal spine.
      c. Stomion moves forward and slightly lower because the upper lip elongates about 1 mm.

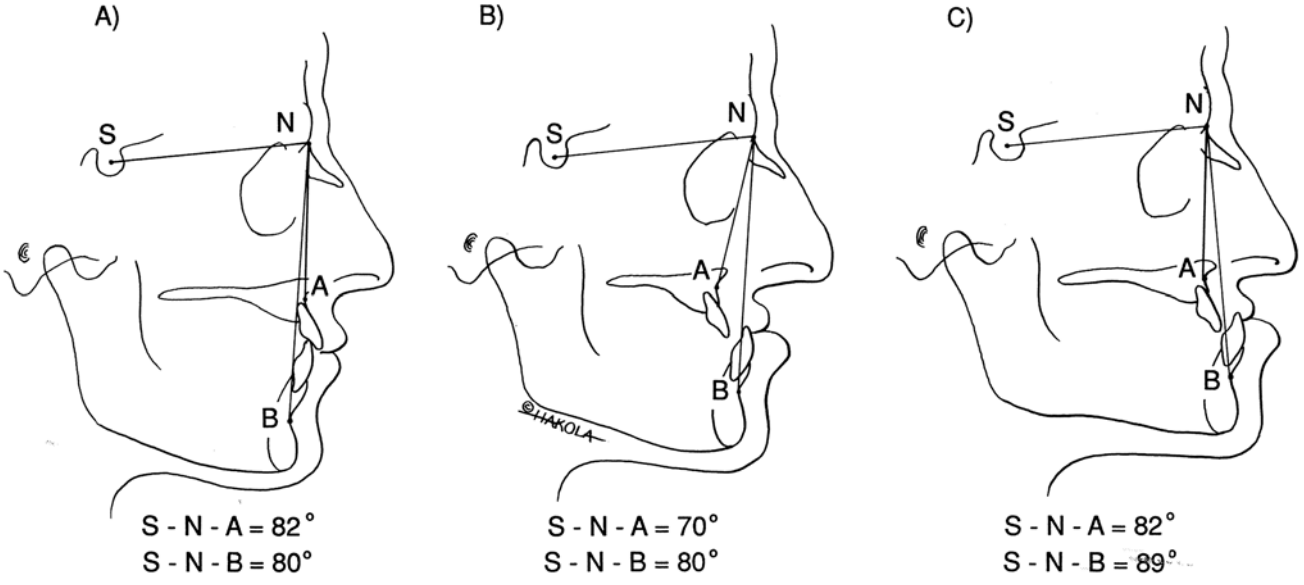

Figure 13.27. (A) Normal. (B) pseudoprognathism. (C) True prognathism.

d. The lower lip is everted. According to Lines,[11] this is variable depending on whether the maxilla is also impacted, in which case this eversion effect will be cancelled.
e. As the maxilla is advanced the upper lip pout is decreased and the vermilion thins. This can be overcome somewhat by the V-Y reapproximation of the upper lip soft tissue as has been reported by Schendel.[12] If the maxilla is impacted the upper lip may shorten by about 2 mm or 20 degrees of the bony impaction. This, too, will be affected by the V-Y closure as described by Schendel.[13]
f. As reported by Carlotti, Aschaffenburg, and Schendel,[13] the most predictable change in the soft tissue of the upper lip is at the vermilion at the level of the central incisor. Here, an advancement of the maxilla and the central incisor of 10 mm results in an advancement of the vermilion by 9 mm. At the level of soft tissue Pt. A, the advancement was 8 mm and at the level of subnasale, 7 mm.
g. The alar base will widen about 10–15 percent of its original width with maxillary impaction and advancement. This can be lessened by an alar base cinch stitch as described by Schendel.[13]

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CHAPTER 14

Prediction Tracing

Stephen A. Schendel

Orthognathic surgery treatment planning is based on a thorough analysis of the patient, cephalometric radiographs, and occlusion, including dental models. Central to surgical planning is the prediction tracing (otherwise known as the visual treatment objective), which is based on the lateral cephalometric radiograph, independent of the type of cephalometric analysis that is used. The prediction tracing permits the surgeon to visualize this treatment objective, therefore allowing refinement of the original plan and ascertainment that the correct soft tissue profile will be obtained for maximum aesthetic value. Construction of the prediction tracing follows in a stepwise fashion. First, the indicated dentoskeletal segments are placed in a class I occlusion and then moved to the position as determined by the facial aesthetic analysis, the cephalometric analysis, and the occlusion. Following this, the relative soft tissue contours in the profile tracing are changed based on known ratios of bone to soft tissue movement. These ratios for specific measurements are outlined in Tables 14.1 and 14.2. Possible skeletal movements include the maxilla (either as a single entity or in segments), the mandible, and the chin. Nasal profile changes, as in rhinoplasty, may also be planned based on this tracing. The visual treatment objective is less accurate for midfacial and craniofacial movements, because visualization and prediction of these soft tissue/skeletal changes is more complex. Thus, its main indication is orthognathic surgery. The prediction tracing will be demonstrated by the following examples.

Case 1 is a cephalometric tracing of a patient with isolated microgenia. All other cephalometric values are normal, and the occlusion is Class 1 with normal dental-to-skeletal base relationships. In Fig. 14.1, the desired level and direction of the osteotomy for an osseous genioplasty has been indicated. This osteotomy should pass at least 5 mm below the canine apex, which is the longest tooth in the anterior region and the main determinant of the vertical position of the osteotomy. The osteotomy must also pass at least 4 mm to 5 mm inferior to the mental foramen. The mental nerve, just prior to exiting the foramen, curves superiorly. Thus, the nerve in the canal is inferior to the actual mental foramen. The isolated mandible and indicated osteotomy segment and relevant soft tissue is shown in Fig. 14.1B. The osteotomized chin segment is then advanced until the menton and pogonion are in the correct cephalometric position and a desirable soft tissue contour of the chin is obtained Fig. 14.1C, D. Generally the movement of soft tissue to bone with an advancement genioplasty is approximately 90 percent. In this case, there has been no major vertical change made in the chin as its vertical position was adequate as determined by the initial cephalometric radiograph. If vertical changes are indicated, they should be done according to the previous plan, either moving the chin segments superiorly by resection of an osseous segment or moving the chin segment inferiorly by downgrafting, followed by the relative soft tissue values being indicated. A chin implant can be inserted into the prediction tracing in a similar manner.

Case 2 is a patient with a vertical maxillary excess, otherwise known as the Long Face Syndrome. The cephalometric tracing has been accomplished, and we can see several salient factors of vertical maxillary excess Fig. 14.2A. One is the excessive show of incisors in relationship to the upper lip. The normal ratio here should be 2 mm to 3 mm based on the measurements of Burstone.[2] The upper lip, when measured from subnasale, is of a normal length; thus, this is not a problem of a short upper lip. There is a concomitant inferior autorotation of the mandible resulting in the long face. The mandible

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Table 14.1. Mandibular procedures.

Type of surgery	Ratio soft/hard tissue
Reduction genioplasty	0.3 : 1
Advancement genioplasty	0.9 : 1
Vertical reduction genioplasty (osteotomy)	0.8 : 1
Mandibular advancement	
chin	1 : 1
labrale inferius	0.75 : 1
Mandibular setback	
chin	1 : 1
labrale inferius	0.9 : 1

Table 14.2. Maxillary procedures.

Type of surgery	Ratio soft/hard tissue
Maxillary advancement	
SN	0.6 : 1
labrale superius	0.9 : 1*
nasal tip	0.27 : 1
Maxillary setback	
SN	0.5 : 1
labrale superius	0.5 : 1
Maxillary impaction	
labrale superius	0.9 : 1*
stomion	0*
Maxillary downgraft LS	0.9 : 1*
stomion	0*

* If V-Y closure and nasal circle are used.

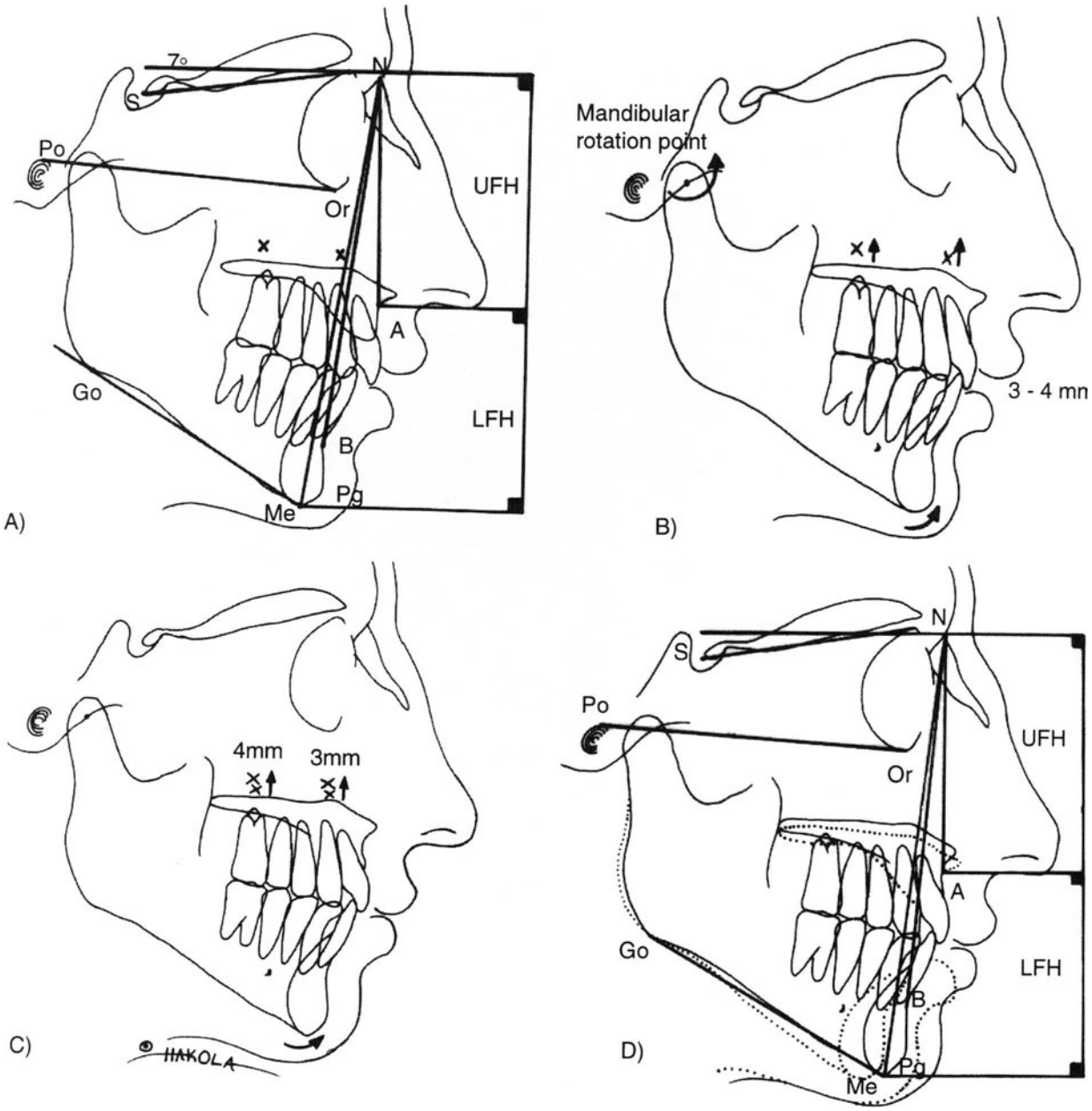

Figure 14.1. Microgenia. (A) Facial plane angle (Po-Or)—(N-Pg). SNA–SNB. Landes Angle (Po-Or)—(N-A). Angle of Convexity (N-A)—(Pg-A). Mandibular Plane Angle (FMA): (Po-Or)—(Go-Me). UFH–LFH. (B) A cut is made up 10 mm from menton back to the 2nd bicuspid. This represents the segment to be cut. (C) Cut segment is advanced to the new position. (D) Final Result.

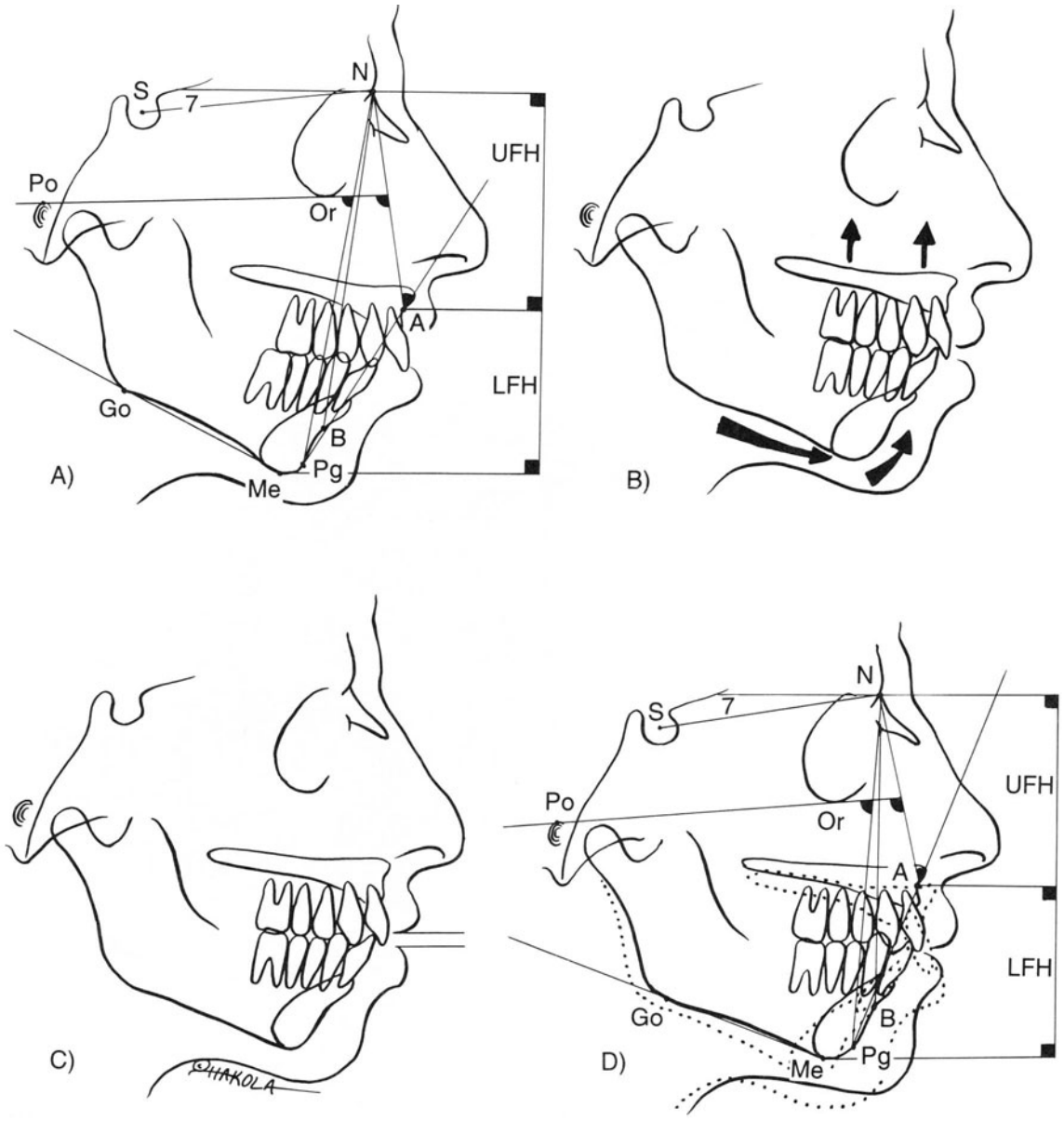

Figure 14.2. Vertical maxillary excess. (A) Facial plane angle (Po-Or)—(N-Pg). SNA–SNB. Landes Angle (Po-Or)—(N-A). Angle of Convexity (N-A)—(Pg-A). Mandibular Plane Angle (FMA): (Po-Or)—(Go-Me). UFH–LFH. (B) As the maxillary vertical dimension is decreased, the maxilla advances slightly and the mandible rotates and advances. (C) Final Occlusion. (D) Result.

is also slightly retrognathic in appearance, with a Class II malocclusion. Cephalometric values in this case would show a slightly decreased SNA angle and an even more decreased SNB angle with an increased ANB angle. Two X's have been placed in the maxillary base area; one over the first molar, and the other just slightly anterior to the canine. The first step in the prediction tracing is seen in Fig. 14.2B. Here, the total mandible, including condyle, has been traced and placed into an ideal Class 1 occlusion with the maxilla, which is then also traced. In cases where presurgical orthodontic treatment has not been completed and is definitely indicated, the axial inclination of the incisor teeth may have to be altered to a more ideal position with the skeletal base. Here, they have already been moved to a normal position by the presurgical orthodontic treatment. Thus, the prediction tracing requires only movement of the skeletal bases to give a normal Class 1 occlusion. With open-bite malocclusions, or some deep-bite malocclusions, a segmental osteotomy of the maxilla or even an anterior subapical osteotomy of the mandible may be indicated, and this should be drawn at this time. Note that the two X's have been transferred to this tracing.

In Step 2. The mandible is placed back into its previous position by superimposing tracing Number 2 over the initial cephalometric tracing. You will note, at this

time, that the maxilla is not in its previous position in tracing Number 1. A point at the center of the condyle is then chosen, and a sharp pencil or compass point is placed here. The maxilla is then rotated in a counterclockwise manner by rotating the mandible-maxillary complex together. This is done until an ideal lip-to-tooth relationship is achieved with the maxillary incisor and the upper lip. Usually 3 mm is a safe, desirable measurement. If a V-Y closure and lip reconstruction is planned at the time of the maxillary osteotomy, no prediction change needs to be made in vertical position for the postoperative lip.[7] If this closure is not done surgically, a 10 to 20 percent shortening of the upper lip should be planned based on the amount of superior movement of the incisor. Once the correct vertical relationship is established, the surgeon then looks at the anterior-posterior relationship of the maxillo-mandibular complex. If the maxilla is in an ideal anterior-posterior position, the final tracing may be done by completing the rest of the drawing, including the cranial base reference point, and by measuring the change in the X's, which will determine the amount of vertical maxillary resection, and will also indicate the direction of AP maxillary movement Fig. 14.2C. If, at this time, the maxilla is determined to be too retrusive or too protrusive, the surgeon then must combine a mandibular osteotomy with the maxillary osteotomy. The maxillo-mandibular complex is thus either advanced or retruded on a horizontal plane. The amount of antero-posterior change then is measured in both the maxilla and mandible. If the patient still has a small chin esthetically. An advancement genioplasty can now be done as shown in the first example. Figure 14.2D shows the completed tracing, using only a maxillary superior impaction and autorotation of the mandible to the desired position.

Prediction tracings for straight maxillary advancement are relatively simple.[4] The maxilla is traced and then advanced into a Class I relationship with the mandible and the new soft tissue profiles are calculated and drawn. In the usual maxillary advancement there will be some tip rise at the nose and the lip will move forward, as indicated in the charts both at the labi superiorus and the subnasal region and these should be calculated in appropriately. If vertical changes are to be made, either shortening or elongation, they are done as in the previous example, rotating the mandible either superiorly or inferiorly if no mandibular osteotomy is to be performed. The planning for mandibular prognatism[5,8,9] and retrusion cases is also relatively straightforward. The mandible is traced except for the posterior segment. It is often helpful to draw out the type of osteotomy in the mandible at this time, whether it be a sagittal, or, for correction of prognathic cases, a vertical oblique osteotomy. The segment proximal to the proposed osteotomy cuts is left untraced. The distal segment of the mandible including the teeth is then either advanced into the proper occlusal relationship with the maxilla, or retruded into the proper position. The stable proximal segment is then drawn and the resultant gap or overlapping area of bone is measured and transferred to the surgery case later on. Following repositioning of the mandible, the chin should be examined for its esthetic position and if any adjustments need to be made here, they are made according to the example in the first figure. Following the movement of the bony segment of the chin, the soft tissues are redrawn.

The prediction tracing is critical to proper and precise treatment planning and surgical execution of the procedure.

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Chapter 15

Genioplasty

Bahman Guyuron

The aesthetic importance of the chin in facial balance and beauty cannot be overemphasized. The labiomental groove, primarily controlled by the position of the chin and teeth, is also an integral component of facial beauty, which has not received the attention it deserves. This chapter reviews pertinent information concerning the chin and the labiomental groove in a systematic fashion.

History

Although it probably was the Egyptians who first conceptualized the aesthetically pleasing face in accordance to the accepted style and trends of their time, it was the Greeks and, later, the leaders of Renaissance movement, which epitomized classical facial features. Michelangelo's concept of beauty was perfectly proportioned units as typified in his famous David. The sculpture has a strong chin with fairly deep labiomental folds, perhaps excessively so by today's aesthetic standards.[1–7]

Early surgical approaches to the chin were designed solely to correct chin deficiencies. Augmentation genioplasty began with the use of many materials such as ivory, gold, silver, and platinum. Subsequent complications ensued, reducing the enthusiasm for these materials.[8] Aufricht reported the use of nasal hump for chin augmentation in 1934.[9]

In 1942, Hofer reported the first osseous genioplasty procedure using a horizontal sliding osteotomy to advance a receding chin through an external anterior chin incision.[10] This cutaneous incision would only have historical significance today.

Eight years later, Converse reported chin augmentation using bone graft through an intraoral incision.[11] In 1952, Gillies used bovine cartilage for chin augmentation.[12] During the same year, Newman reported on genioplasty using dermis graft.[13] Acrylic was probably the first synthetic material used for genioplasty.[14] Silicone implants captivated Brown and Millard in the 1950s, and later Safian.[14–16]

In 1957, Trauner and Obwegeser developed the intraoral approach for a sliding osteotomy of the chin.[17] Proplast was introduced as an augmentation material in 1977.[18] In 1964, Converse discouraged others from contouring the symphysis by removing bone, then he and his colleagues proved the versatility of the anterior horizontal osteotomy of the chin.[19,20]

In 1965, Reichenbach, Kole, and Bruckl described the correction of an elongated lower facial height by resecting a horizontal segment wedged anteriorally.[21]

Correction of the "witch's chin" deformity with an external triangular skin excision was first described by Gonzales-Ulloa in 1972, while Loeb in 1978 described a method with submental fat flap to correct this undesirable chin shape.[22,23]

Hydroxyapatite as an inlay for genioplasty was first reported by Zeller in 1986.[24]

For the anatomy of the chin area and patient evaluation, the reader is referred to the previous chapters in this book.

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Table 15.1. Soft tissue response to bone adjustment.

Augmentation	
0.80 to	0.90
1.00	1.00
Ostectomy	
Horizontal	0.25
	1.00
Vertical	0.50
	1.00
Osteotomy	
Retraction	0.90
	1.00
Vertical reduction	0.80
	1.00
Advancement	0.90
	1.00

Figure 15.1. Riedel's plane is a line that connects the most prominent portion of the upper and lower lips and touches the soft tissue pogonion (the most prominent anterior portion of the chin on an ideal balanced face).

The Role of Life-Size Photographs and Cephaloxerograms

A set of life-size front view and profile photographs of the patient is obtained and analyzed. These are the photographs that are taken with an indicator or ruler on the side of the face at the level of the lateral canthus on front view and at the level of the nasal dorsum on the profile. The photographs are then enlarged by the photographer to an exact and normal facial size by comparing the measurement of the indicator or ruler to the actual size of the indicator in the darkroom.

Analysis of the front view, which will exhibit any anterior facial height deficiency or excess, combined with a simple analysis and measurement in the profile view, will define the deformity and help to plan the corrective procedure.

The combination of front view and profile analysis will provide a blueprint of the surgery with the difference between the ideal chin shape and position and the existing chin outline expressed in millimeters. Having this information and knowledge of the soft tissue response to the skeletal alteration (Table 15.1), one can precisely perform a genioplasty, regardless of whether it is an augmentation or osseous procedure.[25,26] Discussing the numerous techniques of analysis of the photographs or cephalometric x-rays of the lower face in relation to genioplasty exceeds the scope of this chapter.

Providing the lip anatomy is correct, the most useful single profile analysis is Riedel's plane (Fig. 15.1). In this analysis, the line that connects the most protruding portion of the upper and lower lips touches the most prominent anterior portion of the chin (pogonion) in an ideal position. Should the chin fall posterior to this line, the gap between pogonion and Riedel's line, measured in millimeters, exhibits the degree of deficiency. If this line passes posterior to pogonion, the chin is excessive, which is also measured in millimeters.

The front view analysis can help in the detection of excess or deficiency of the lower facial height. The distance from stomion to menton should be twice as long as the length of the upper lip. The latter is measured as the distance from stomion to subnasale. The lower facial height also equals the distance from subnasale to the medial canthus line. If the distance from stomion to menton is short, then the patient has vertical microgenia. If it is long, the patient has vertical macrogenia providing that the other facial structures are normal.[27]

Table 15.2. Classification of chin deformities.

Group I:	Macrogenia Horizontal Vertical Combination
Group II:	Microgenia Horizontal Vertical Combination
Group III:	Combined micro and macrogenia Vertical excess and horizontal deficiency Vertical deficiency and horizontal excess
Group IV:	Asymmetric chin Normal anterior lower facial height Short anterior lower facial height Long anterior lower facial height
Group V:	Pseudomacrogenia
Group VI:	Pseudomicrogenia
Group VII:	Witch's chin deformity

Cephaloxerograms and orthopanograms are helpful in detecting the degree of soft tissue excess and any skeletal abnormalities often missed during the initial examination.

Classification of Chin Deformities

As is shown in Table 15.2, there are seven categories of chin deformities.[28] The type of deformity will be a guide to the selection of the appropriate surgical approach.

The first category of deformities, macrogenia, can be horizontal, vertical, or a combination of both (Fig. 15.2). Pure horizontal macrogenia can seldom be successfully corrected with an ostectomy (shaving down the bone), because the soft tissue response to the skeletal alteration is small and unpredictable. These patients are more appropriately served by setting the chin bone back, even though occasionally ptosis of the soft tissue in the submental area can be an undesirable outcome of this surgery (Fig. 15.2A–C).

Correction of pure vertical chin excess, however, is more predictably corrected by resecting a horizontal segment in the shape of a block or wedge (Fig. 15.2B). Soft tissue response is more reliable and the outcome of surgery is more desirable. Combination of horizontal and vertical macrogenia can only be successful with an osteotomy, resection of a horizontal segment, and a setback (Fig. 15.2C).

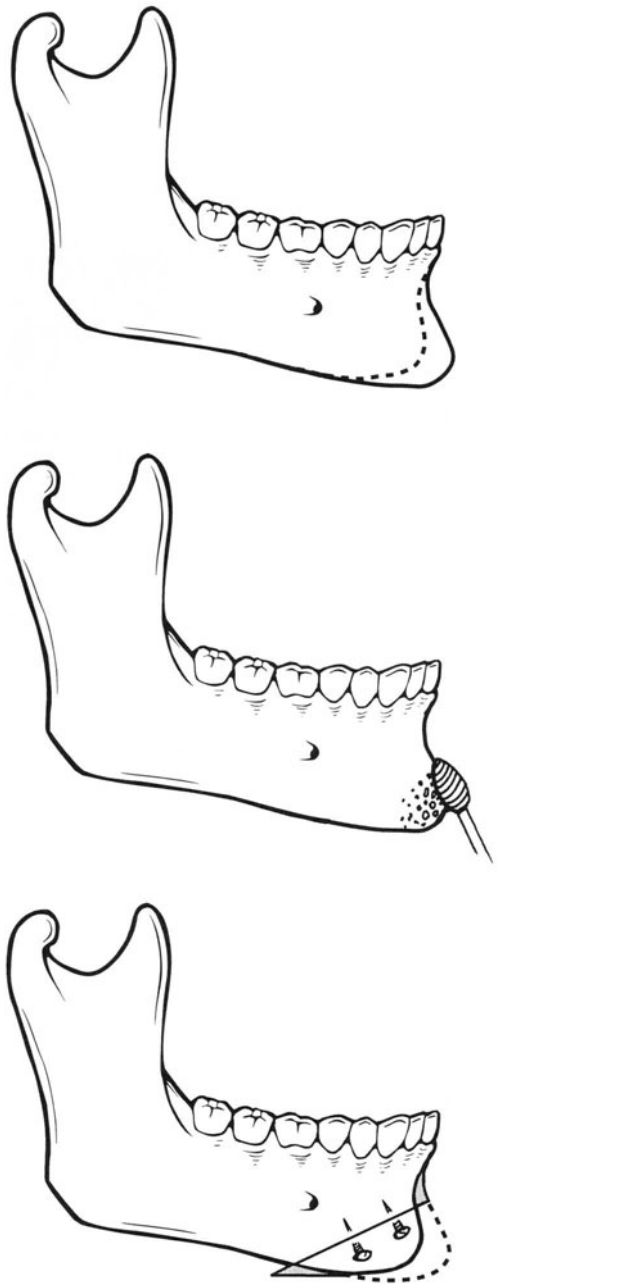


Figure 15.2. Macrogenia. (A) 1. Artistic illustration of horizontal macrogenia with the broken line showing the normal chin bone outline. 2. Artistic illustration of ostectomy with an oval burr, one choice for correction of horizontal macrogenia. 3. Two oblique osteotomies, with resection of a segment setting the caudal segment back, will have a more successful effect on horizontal macrogenia.

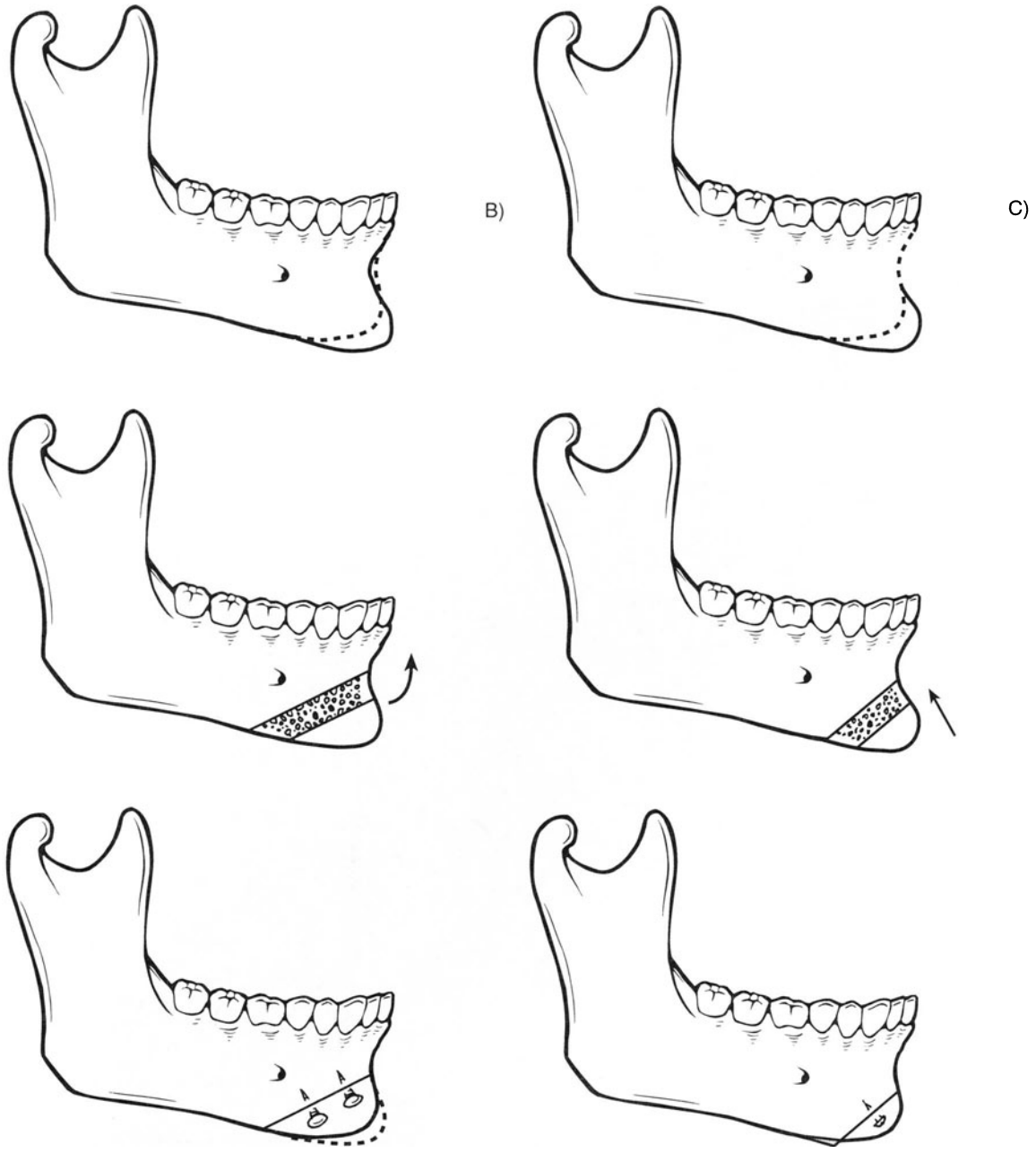


Figure 15.2. *Continued.* (B) 1. Artistic illustration of vertical macrogenia with the broken line outlining an almost horizontally oriented normal chin bone. 2. The design of two osteotomies to shorten the facial height. 3. The caudal segment is moved cephalad and fixed in position using two screws. (C) 1. Illustration of combination macrogenia with the broken line representing the normal chin outline. 2. Design of two obliquely oriented osteotomies to reduce the chin vertically and horizontally. 3. The excess segment is removed and the remaining segment is moved to an appropriate position and fixed with screws.

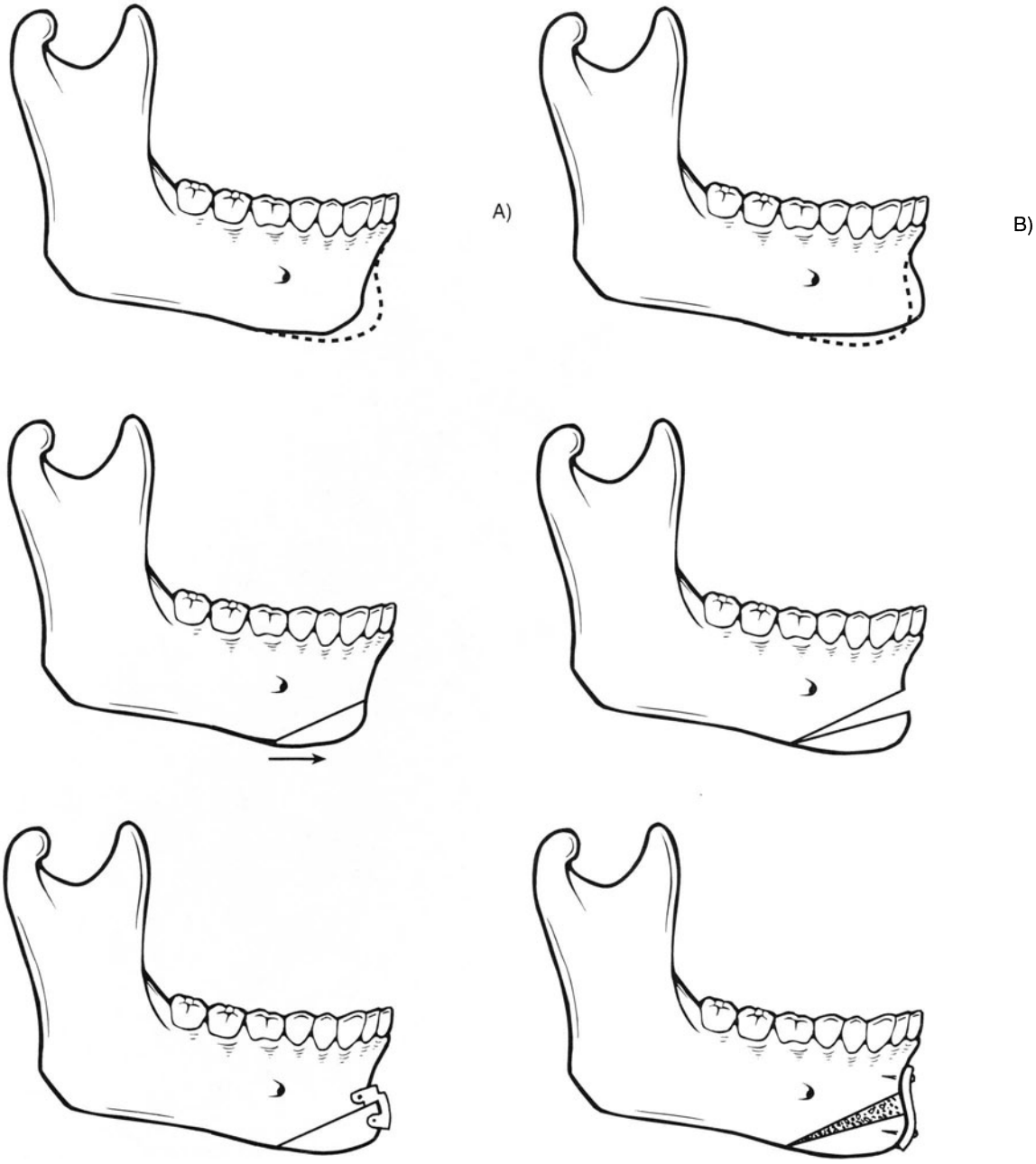
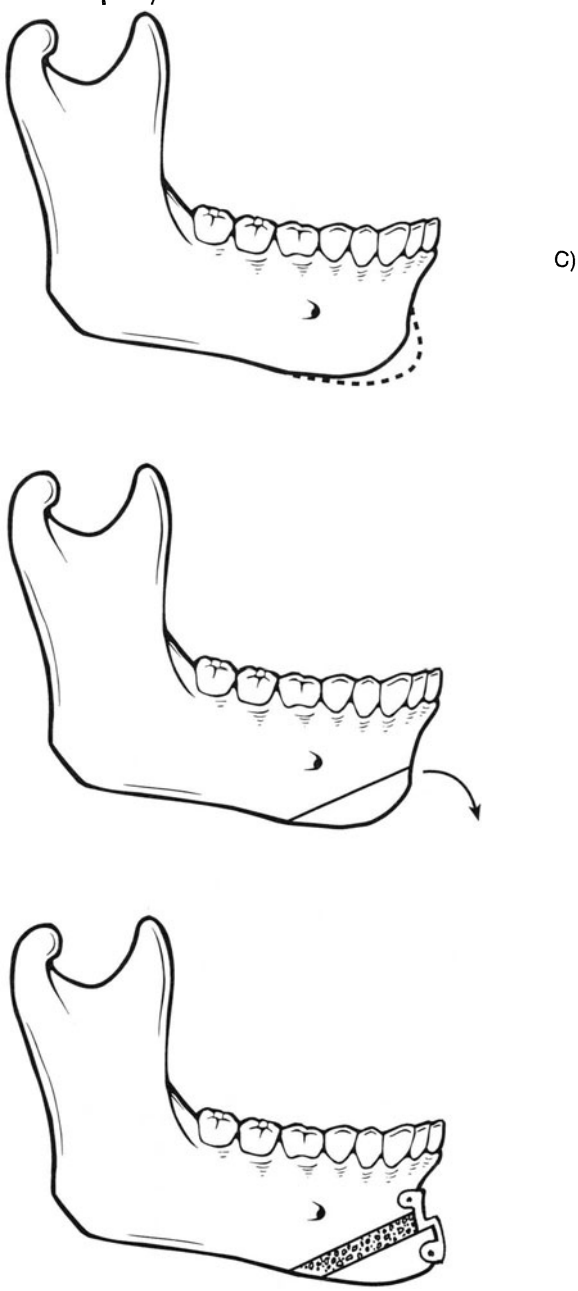


Figure 15.3. (A) 1. Artistic illustration of horizontal microgenia with the normal chin line depicted with a broken line. 2. Design of a horizontal osteotomy to advance the caudal segment. 3. The caudal segment is advanced and fixed in position using plates and screws. (B) 1. Artistic illustration of vertical microgenia with the broken line showing the normal chin bone outline. 2. Design of an osteotomy to correct the vertical deficiency. 3. The caudal segment is moved caudally and fixed in position with interposition of bone graft or hydroxyapatite.

Microgenia, the most common type of chin deformity, is subdivided into horizontal, vertical, or a combination of vertical and horizontal microgenia (Figs. 15.3, 15.4, 15.5). The candidates for correction of microgenia with an implant are patients with an isolated horizontal chin deficiency, where the addition of an implant to the anterior symphysis can produce a fairly predictable result. A combination of horizontal and vertical deficiency or vertical deficiency alone cannot be corrected as successfully with an implant. Generally, patients with this type of deformity benefit more from an osteotomy and interposition bone graft or hydroxyapatite (Fig. 15.3C).



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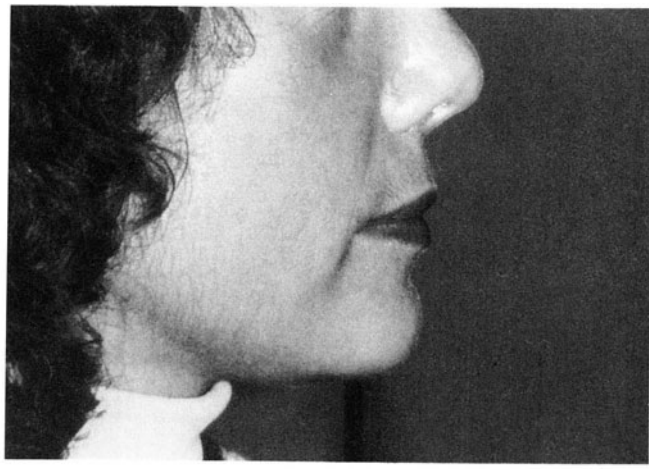

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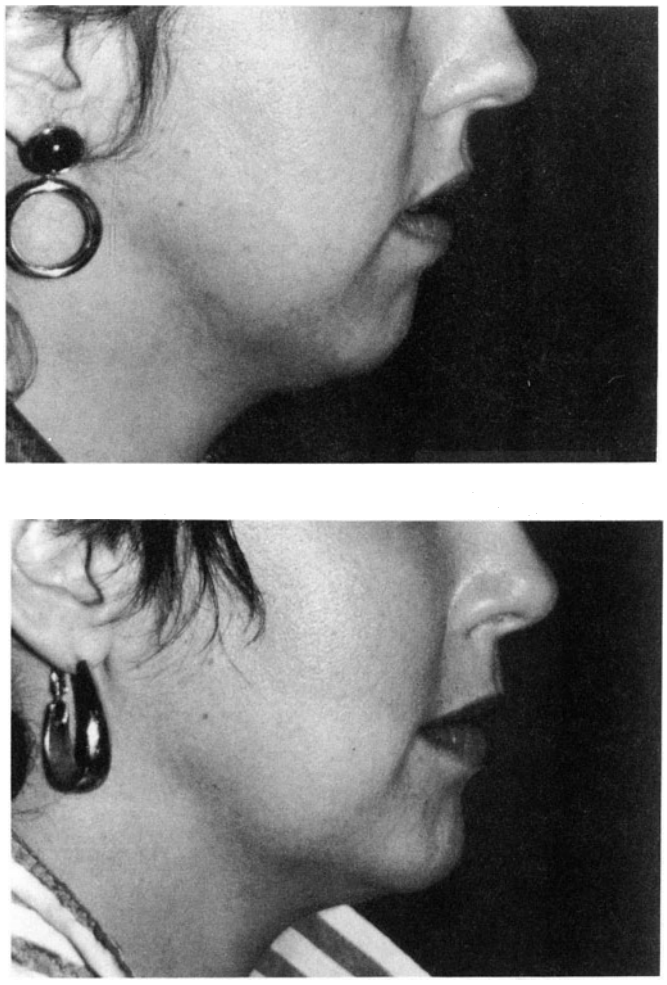
Figure 15.3. *Continued.* (C) 1. Illustration of combination vertical and horizontal microgenia, with the normal chin outline shown in broken lines. 2. Design of a horizontal osteotomy. 3. The caudal segment is moved caudally and anteriorly, fixed in position and bone or hydroxyapatite is placed in the resultant gap.

Figure 15.4. Horizontal microgenia (A), whose deformity is corrected with advancement genioplasty and rigid fixation (B).

The third group of chin deformities are those patients who have excess bone in one direction and deficiency in another. These can either be horizontal deficiency with vertical excess or horizontal excess with vertical deficiency (Fig. 15.6). Both of these categories are improved with an osteotomy and reduction in one direction and an increase in the other direction.

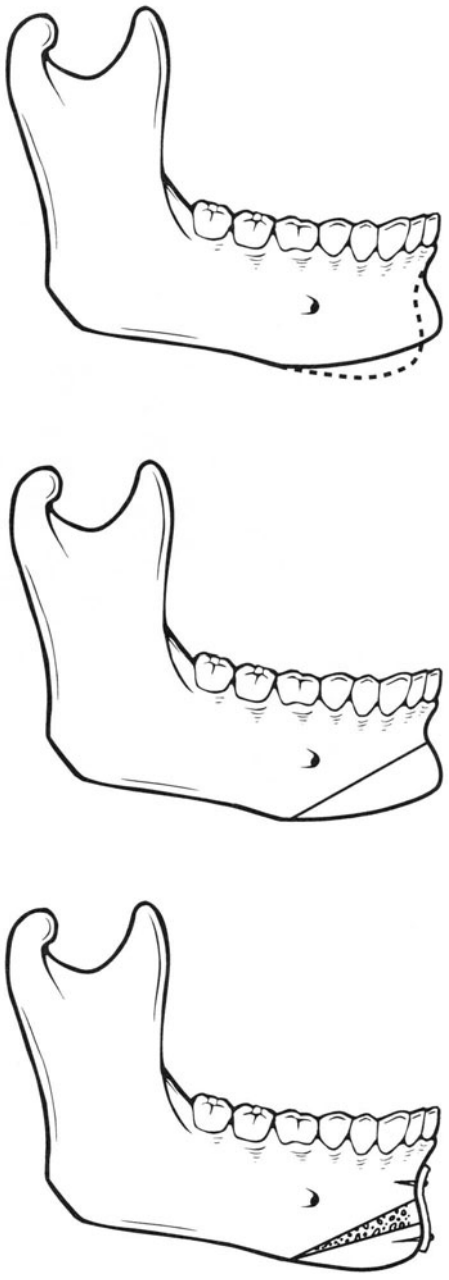
The fourth group of chin deformities are asymmetric chins (Fig. 15.7). These are also more effectively corrected using an osteotomy rather than augmentation.



A)

B)

FIGURE 15.5. Preoperative photograph of patient with horizontal microgenia, minor nasal imperfection, and lip incompetence (A). The same patient following advancement genioplasty and rhinoplasty (B).



A)

FIGURE 15.6. Combined horizontal microgenia and vertical macrogenia. (A) 1. Artistic illustration of vertical deficiency and horizontal excess with the normal chin bone outline represented by the broken lines. 2. The design of the proposed osteotomy. 3. The caudal segment is moved caudally and posteriorly. Whether bone or bone substitute will be necessary to fill the gap, is dependent upon the degree of caudal advancement.

Asymmetric chins are subdivided into three categories depending on the lower anterior facial height. For an asymmetric chin with normal facial height, a wedge should be removed from the excess side and added to the short side to allow rotation of the chin to a normal position (Fig. 15.7A). If, however, an asymmetric chin exists with excess anterior facial height, a segment is removed from the long side of the chin and discarded. The caudal segment is moved cephalad, the midline is lined up, and the segment is fixed in position (Fig. 15.7C). Should the chin be vertically short, yet asymmetric, a horizontal osteotomy is made and the segment is moved to a proper position creating a normal anterior facial height (Fig. 15.7B). The resultant gap between the segments is then reconstructed with bone graft or hydroxyapatite (Table 15.3). The combination of chin asymmetry with horizontal chin deficiency or excess will be an even less likely condition.

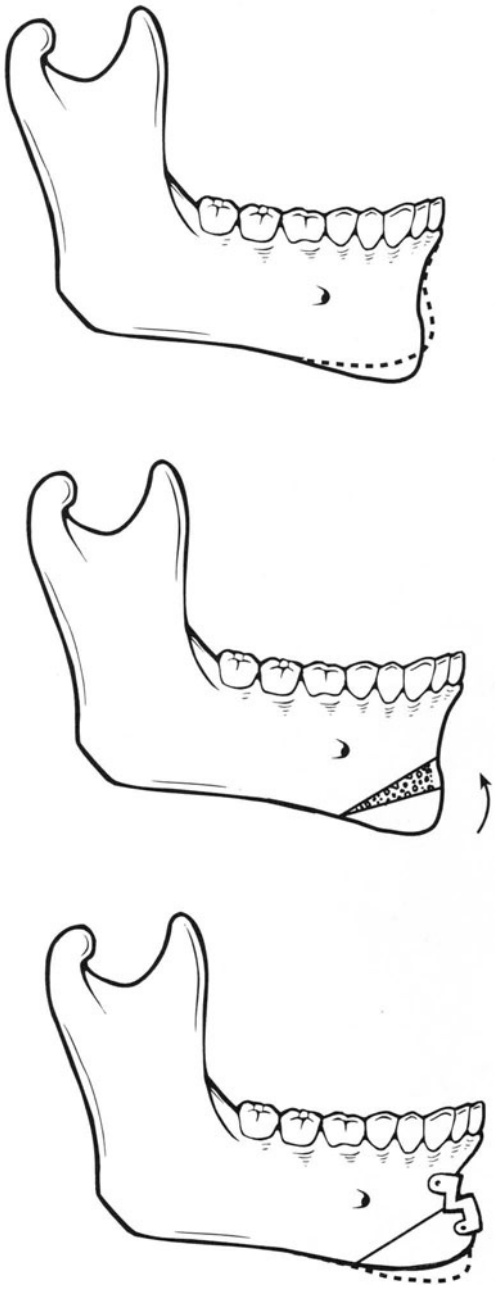


FIGURE 15.6. *Continued.* (B) 1. Artistic illustration of vertical excess and horizontal deficiency. The broken line represents the normal chin bone outline. 2. The design of osteotomies to correct the deformity. 3. The excess portion of the bone is removed and the segment is advanced anteriorly.

The fifth group of chin deformities are those we have called pseudomacrogenia. These are the patients who have excess soft tissue of the chin resulting in an appearance of macrogenia while the chin bone has a normal shape and position (Fig. 15.8). These patients will require soft tissue adjustment, which is difficult and unpredictable.

Patients with pseudomicrogenia are those who have had excessive growth of the maxilla, resulting in clockwise rotation of the mandible and chin. This creates an illusion of microgenia, while cephalometric analysis reveals a normal lower face. As the long face deformity is corrected by intrusion of the maxilla, the chin will rotate anteriorly and regain its normal position.

The final category of chin deformities is "witch's chin," a soft tissue ptosis, resulting in an undesirable appearance. These are patients who would benefit from a correction of the deformity using techniques that would alter the soft tissue.[28–35]

Augmentation Genioplasty

As already stated, the patients who have pure horizontal deficiency of the chin are reasonable candidates for augmentation genioplasty. Those who have vertical deficiency rarely benefit from this technique. Those of us who are trained in craniofacial surgery do not advocate the liberal use of any alloplastic substance for augmentation and, therefore, choose this approach only on a limited basis, as for those patients with minimum deficiency and in an older age group. Use of autogenous material for chin augmentation extends the indication to younger patients and those with greater chin deficiencies.

These patients are evaluated in the manner described. The life-size photographs will indicate the degree of chin deficiency expressed in millimeters. The ratio of soft tissue response to the augmentation genioplasty is 0.8 to 1. Therefore, a 5 mm chin deficiency would be corrected using a 6-mm thick implant or bone or cartilage graft.

Materials for Augmentation Genioplasty

A large number of materials have been used for augmentation genioplasty (Table 15.3). However, of these

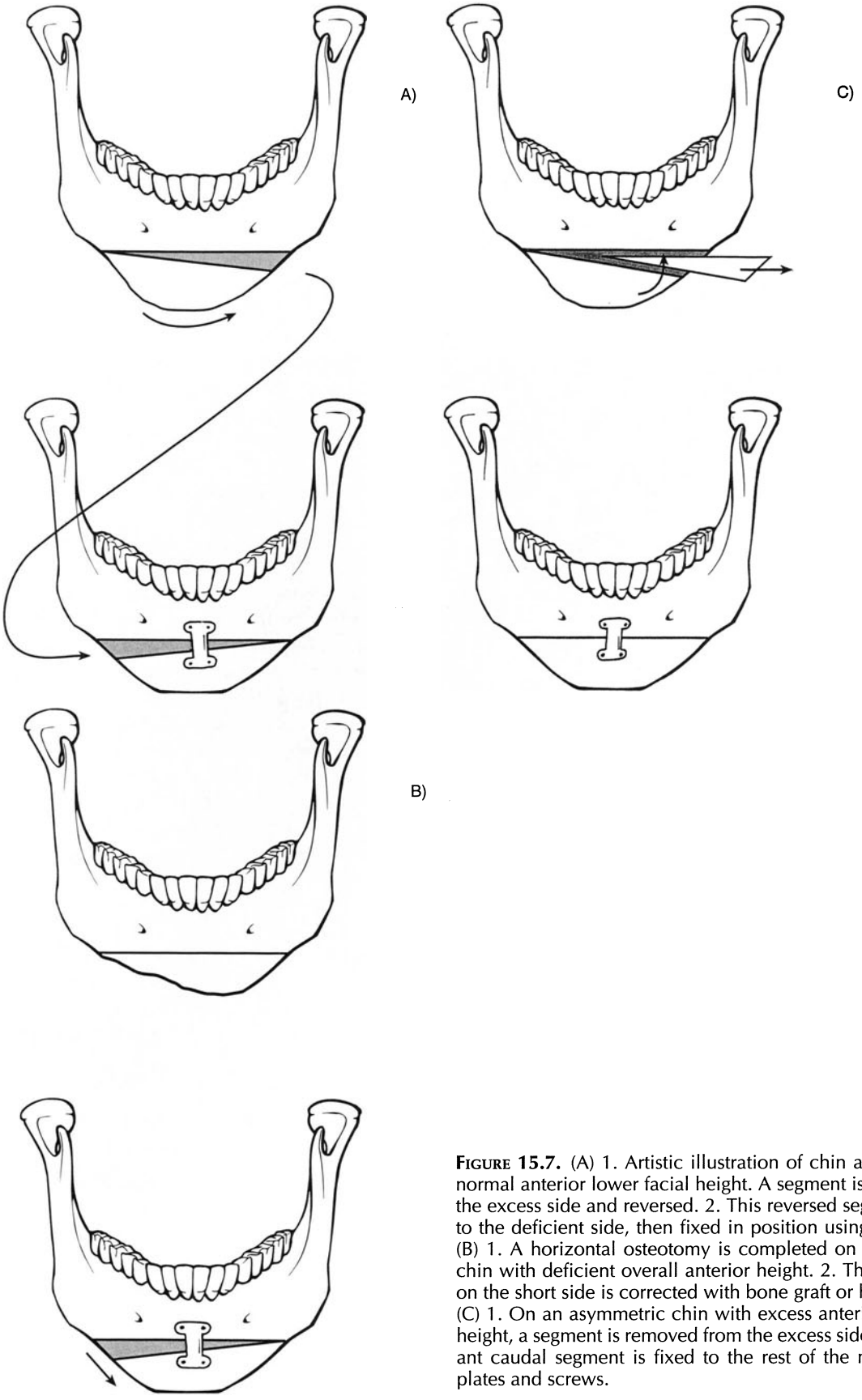


Figure 15.7. (A) 1. Artistic illustration of chin asymmetry with normal anterior lower facial height. A segment is removed from the excess side and reversed. 2. This reversed segment is added to the deficient side, then fixed in position using rigid fixation. (B) 1. A horizontal osteotomy is completed on an asymmetric chin with deficient overall anterior height. 2. The gap resulting on the short side is corrected with bone graft or hydroxyapatite. (C) 1. On an asymmetric chin with excess anterior lower facial height, a segment is removed from the excess side. 2. The resultant caudal segment is fixed to the rest of the mandible using plates and screws.

Table 15.3. Materials used for augmentation genioplasty.

Paraffin injection	Methylmethacrylate
Ivory	Nasal hump
Gold	Marlex 50
Silver	Polyethylene
Platinum	Solid polyethylene
Iliac bone	Polyvinyl sponge
Rib cartilage	Silicone
Homologous graft	Proplast
Diced cartilage	Hydroxyapatite
Submental fat	

materials, only a few have clinical value in today's practice. These can be divided into autogenous, such as bone and cartilage graft, dermis grafts, or alloplastic, which primarily includes silicone, hydroxyapatite, Proplast* and methylmethacrylate.

Proplast, which is not currently available, was a popular material because of its porous nature and slight malleability. It was available in a size range from 4 to 12 mm. Silicone is nonporous and available in different sizes and configurations. However, silicone implants do produce a good deal of capsular contracture, which if the implant is placed in the supraperiosteal plane, can result in an abnormal chin appearance.

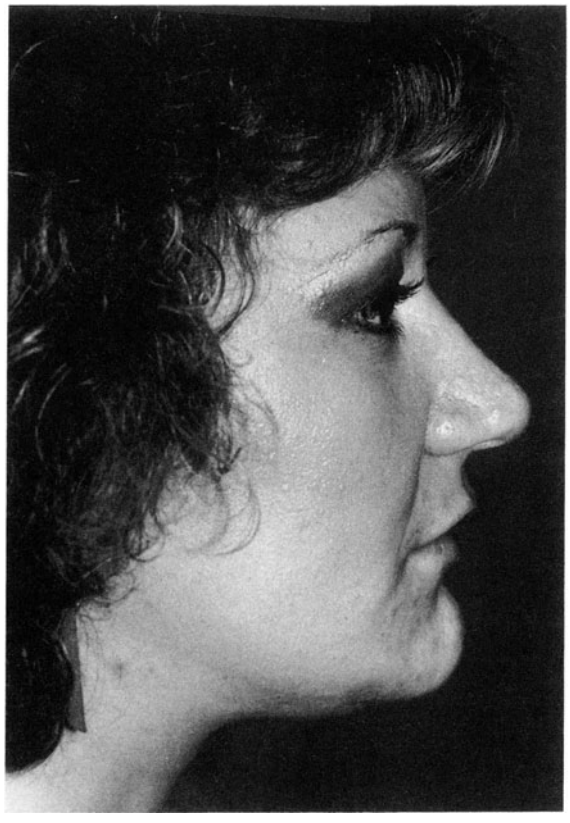


Figure 15.8. Excess soft tissue causes the appearance of macrogenia in this pseudomacrogenia example.

Hydroxyapatite, although one of the more ideal materials biologically, is not suitable for genioplasty due to the unavailability of properly shaped implants, difficult in contouring, and impossibility of adapting the shape of the underlying bones. A significant effort by several researchers, including the author, has been invested recently in the development of hydroxyapatite and methylmethacrylate derivitive implants such as HTR.† More anatomical shapes of these materials are now available (Fig. 15.10F and 15.10G).

Autogenous materials have the distinct disadvantage of requiring an incision to harvest the graft. Unless this procedure is being done in concomitance with another operation, which requires harvesting bone graft, one cannot readily justify harvesting bone merely for the sake of augmentation genioplasty, except in unusual cases. Other disadvantages of autogenous augmentation genioplasty are donor site morbidity and scars.

Technique

An augmentation genioplasty can usually be performed under local anesthesia with intravenous sedation. We currently use a combination of propofol,‡ midazolam hydrochloride,§ and fentanyl.‖ The face is first prepped and draped. If the intraoral approach is selected, the lower labial sulcus is infiltrated with lidocaine hydrochloride containing 1:200 000 epinephrine to achieve a bilateral mental nerve block. Time is allowed for the effect of the epinephrine. An incision is then made on the labial side, 6–8 mm from the labial sulcus to avoid gum retraction and, ultimately, root exposure (Fig. 15.9A). An adequate cuff of soft tissue is left for a waterproof repair. Gingiva retraction with tooth root exposure and dehiscence are disastrous and of significant consequences.

The incision is then deepened toward the bone and the periosteum is elevated (Fig. 15.9B and 15.9C). A pocket is created, only large enough to accommodate the implant. The implant is then impregnated with an antibiotic containing solution and the cavity is irrigated with the same solution. The implant is lined up symmetrically with the facial midline. Chromic catgut (4-0) is used to repair the muscle, and 4-0 and 5-0 chromic catgut are used to repair the submucosa and mucosa layers (Fig. 15.9D). If the pocket is dissected adequately, fixation of the implant is not always necessary. Should there be any question concerning migration of the implant, it should then be fixed in position with two screws or a nonabsorbable suture.

* Proplast, NovaMed, Inc., Houston, TX.
† HTR, Poly-Medics, Warsaw, IN.
‡ Deprivan, Stuart Pharmaceuticals, Wilmington, DE.
§ Versed, Roche Laboratories, Little Falls, NJ.
‖ Sublimaze, Janssen Pharmaceutica, Piscataway, NJ.

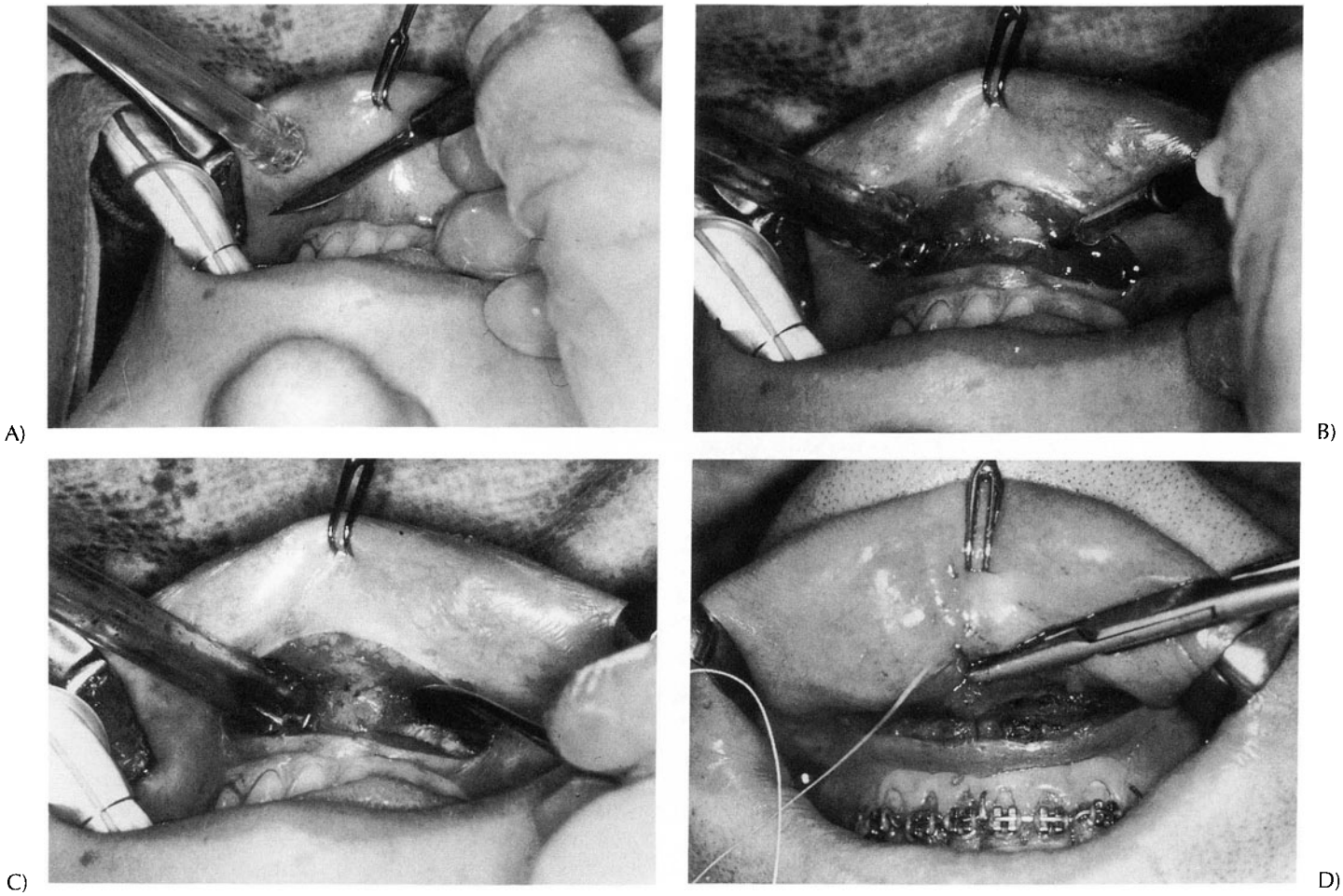


Figure 15.9. (A) The incision is made about 8 mm on the lip side of the lower labial sulcus using a 10 blade to cut the mucosa. (B) The remaining soft tissue and periosteum are incised using the cutting power of a cautery. (C) A periosteal elevator creates the appropriately sized space for the implant over the symphysis without excessive undermining cephalad. (D) The mentalis muscle is reattached with 4-0 polyglactin 910 (Vicryl) or chromic catgut and the mucosa and submucosal planes are repaired with 4-0 chromic catgut.

Should the submental incision be selected, which is the preferred approach to an alloplastic genioplasty, the cutaneous midline of the chin is marked and a horizontal incision is designed just anterior to the existing submental fold, if indeed it does exist. The area is then infiltrated with lidocaine hydrochloride containing 1:200000 epinephrine, including the area of the mental foramen to achieve a nerve block. Because proper hemostasis is an integral part of a successful augmentation genioplasty, direct injection of the chin is carried out in addition to the nerve block. The skin incision is then made and taken through the subcutaneous tissue down to the periosteum (Fig. 15.10A and 15.10B). The periosteum over the anterior symphysis is elevated and a pocket is created (Fig. 15.10C, 15.10D, and 15.10E). The midline mark is then transferred to the bone, the appropriate chin implant size is selected, and the midline of the implant is also marked (Fig. 15.10F). The periosteum is elevated and the chin implant is placed in position (Fig. 15.10G and 15.10H). Again, the implant can be fixed using one or two screws, but often this is not necessary. When the pocket is only dissected large enough to accommodate the implant in an ideal position, fixation may not be mandatory. The periosteum is then repaired using 5-0 polyglactin 910.[#] The subcutaneous soft tissue is also repaired in layers using 6-0 polyglactin 910 and finally, the skin is approximated using 6-0 plain catgut. Throughout the process, the wound and the implant are irrigated with an antibiotic solution. A pressure chin strap, either using a Jobst type garment or adhesive foamy sponge tape, is applied.

Should the intraoral incision be used, the patient resumes a normal diet the next day, staying on a liquid diet the first day. The submental approach does not require any special diet. A first generation cephalosporin is prescribed for 5 days.

Advantages of Augmentation Genioplasty

Its simplicity constitutes the biggest advantage of augmentation genioplasty. This operation can usually be done in 15 to 20 minutes. The patients who undergo this procedure experience less pain, swelling, and paresthesia postoperatively, as compared to an osteotomy, although this has not been statistically substantiated.[34]

[#] Vicryl, Ethicon, Inc., Somerville, NJ.

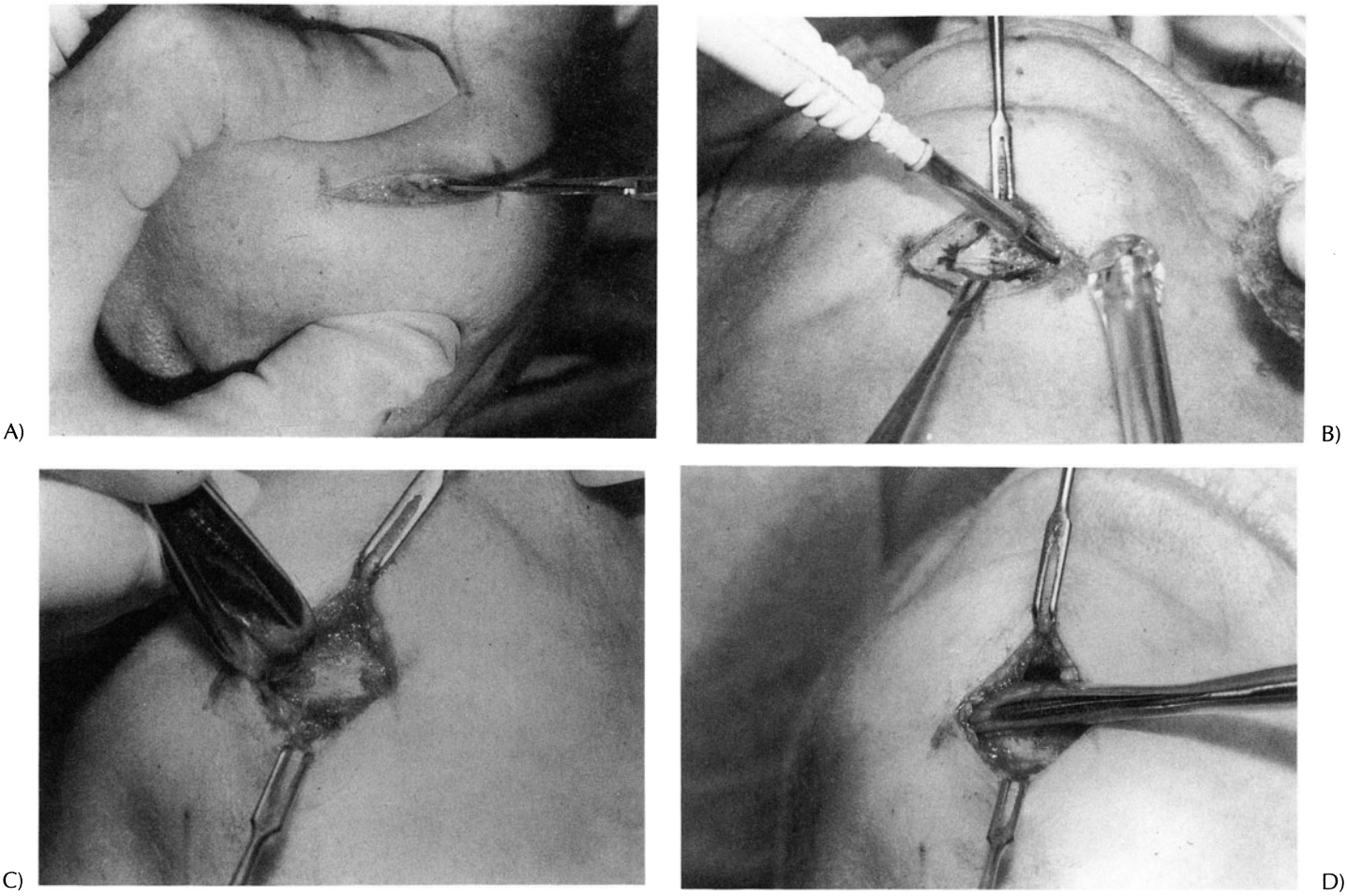


FIGURE 15.10. (A) A cutaneous incision is placed anterior to the existing submental fold following infiltration of the area with a local anesthesia. (B) An electrocautery deepens the incision. (C) Once the subperiosteal plane is entered, the periosteum is elevated centrally. (D) The periosteum is elevated on the right side.

The procedure is reversible, but might result in soft tissue chin deformity. It is almost always possible to perform this procedure under local anesthesia with or without intravenous sedation.

Disadvantages of Augmentation Genioplasty

The patients who undergo augmentation genioplasty using alloplastic material have a slightly higher possibility of infection. Although rejection has never been documented, it is one of the theoretical disadvantages that one should consider. Bone resorption under the implant is common, more so if the implant is placed subperiosteally.[25] Implant migration is a possibility if the pocket is larger than the implant and a fixation device is not used. As long as the prepared site is dissected symmetrically and just large enough to host the implant, displacement can be avoided. Capsular contracture is a distinct disadvantage of silicone implants. Soft tissue response is less predictable with augmentation genioplasty. Twenty percent of the patients are also aware of the implant's presence on a continuous basis.[35]

Another distinct disadvantage of augmentation genioplasty is its limitation in correcting different classes of chin deformities. Only patients with microgenia benefit from this procedure.

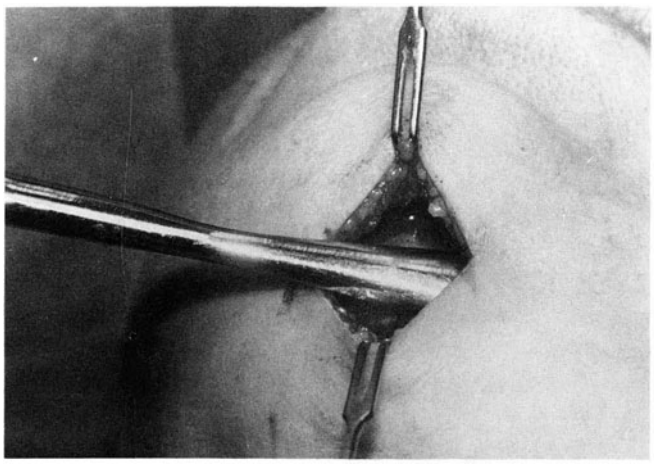
Complications

Complications of augmentation genioplasty using alloplastic material include infection, rejection, capsular contracture, wound dehiscence, permanent paresthesia, and dental root erosion. These are very unlikely. Wound dehiscence usually results from not leaving an adequate amount of soft tissue on the gingival side of the incision for an appropriate repair.

Use of alloplastic material should be avoided in patients with diabetes, periodontal disease, significant chin deficiencies, any type of chronic infection, and immunosuppression. Also, patients in a younger age group should not undergo augmentation genioplasty unless there is a compelling reason.

Osseous Genioplasty

As stated earlier, osteoplastic genioplasty is the most versatile procedure available for correction of chin deformities. This procedure can be divided into two cate-



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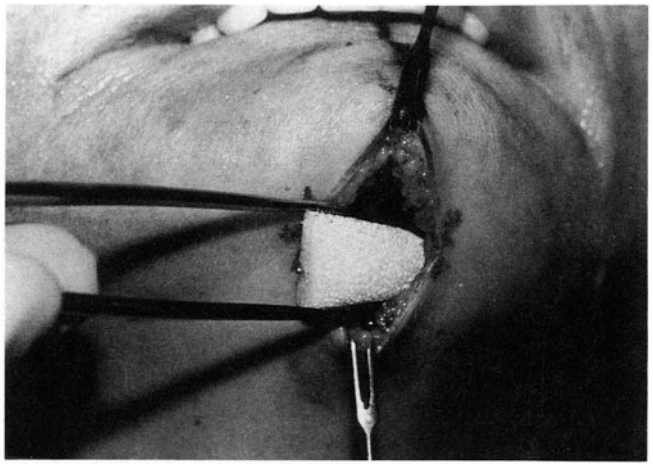

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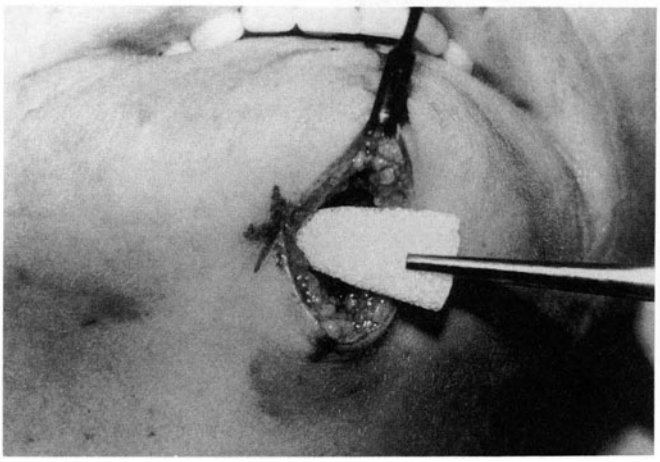


G)

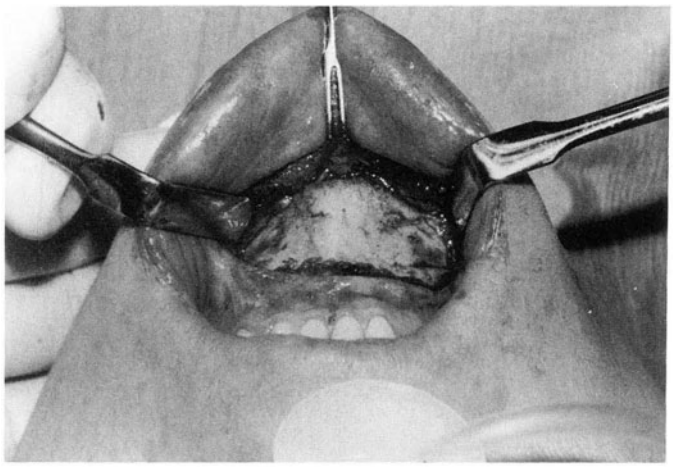
H)

FIGURE 15.10. *Continued.* (E) The periosteum is elevated on the left side. (F) The midline of the implant is marked to be matched to the midline of the chin. (G) The implant is being introduced into the space, one side at a time. (H) The midline of the implant is aligned with the midline of the existing chin.

gories. Ostectomy, which means removing the bone using a burr, and osteotomy.

Ostectomy

This technique has limited application and can only be used in patients with isolated excess of the anterior symphysis or, as stated previously, horizontal macrogenia. The initial part of the procedure is similar to an intraoral augmentation genioplasty. The periosteum is elevated until the symphysis is exposed completely (Fig. 15.11A). The dissection is slightly more extensive and the mental foramina are exposed to protect the nerve. Next, the midline is marked vertically and a large oval burr is used to remove the bone, each side individually (Fig. 15.11B and 15.11C). After one half is resected, the depth of the planed area can be measured with a caliper, so the surgeon has a clear idea of the amount of bone being removed. The other side is then gently removed until it is evenly leveled with the side reduced first (Fig. 15.11D). The wound is then irrigated and a detailed repair of the layers is accomplished with 4-0 chromic catgut (Fig. 15.11E). The soft tissue response to ostectomy is extremely poor and as stated earlier, very unpredictable (Table 15.1).



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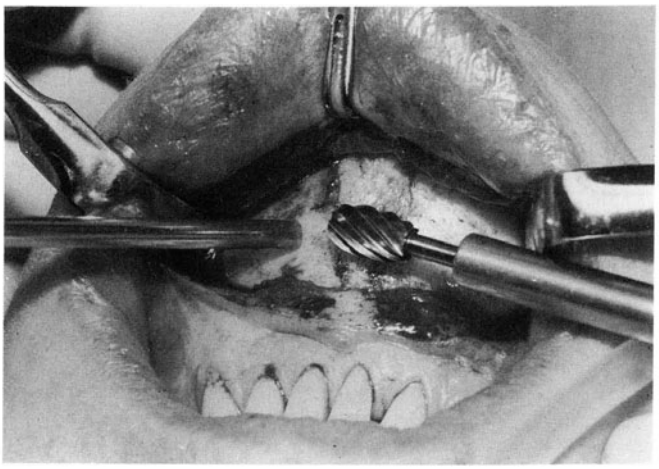

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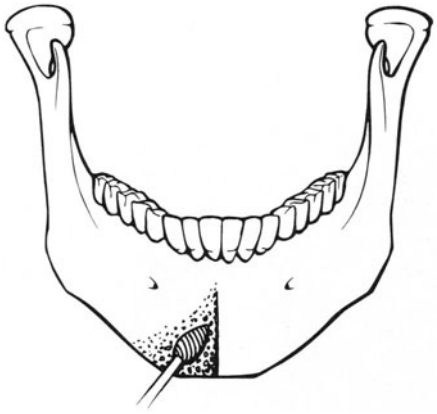

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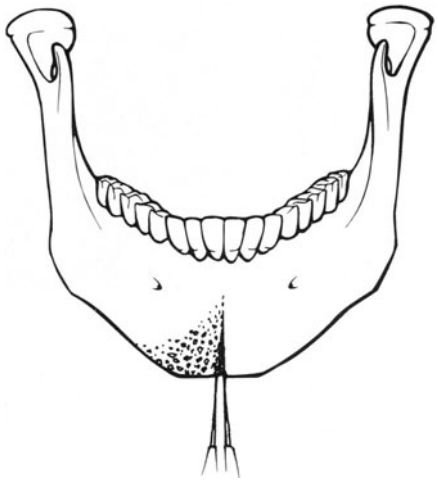

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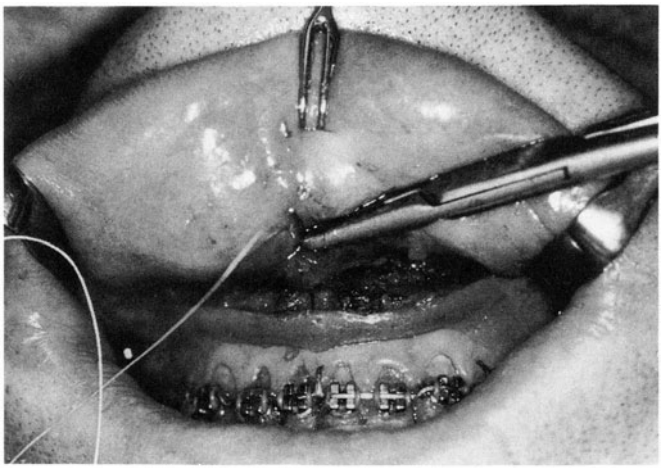
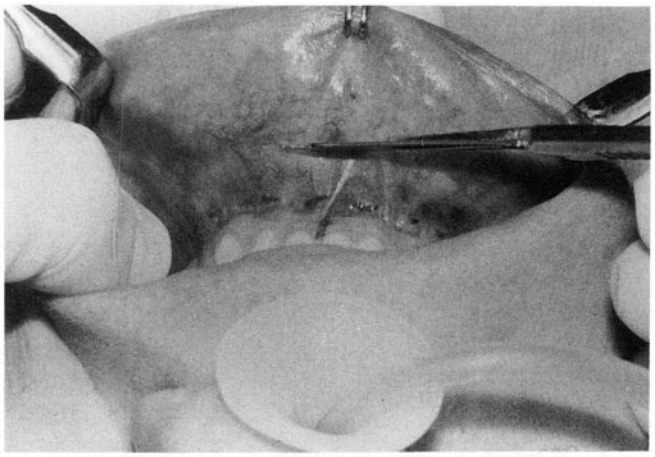


E)

FIGURE 15.11. (A) The soft tissue is dissected down to the periosteum, which is then elevated and retracted. (B) An ostectomy being completed with a large oval burr removes bone starting from the midline. (C) Artistic illustration of bone removal using the oval burr in a tapered fashion, one side at a time. (D) The depth of the resected chin bone can be calculated using the method in this illustration. After a satisfactory reduction is done on one side, the other side is leveled evenly. (E) 4-0 polyglactin 910 (Vicryl) or chromic catgut is used the reattach the mentalis muscle, then 4-0 chromic catgut is used to repair the oral lining.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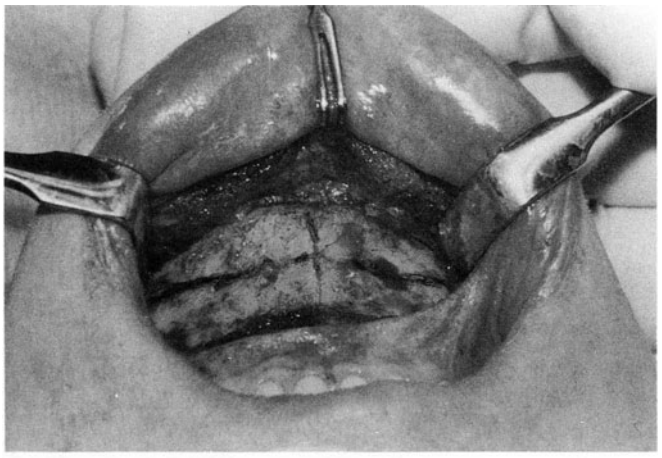
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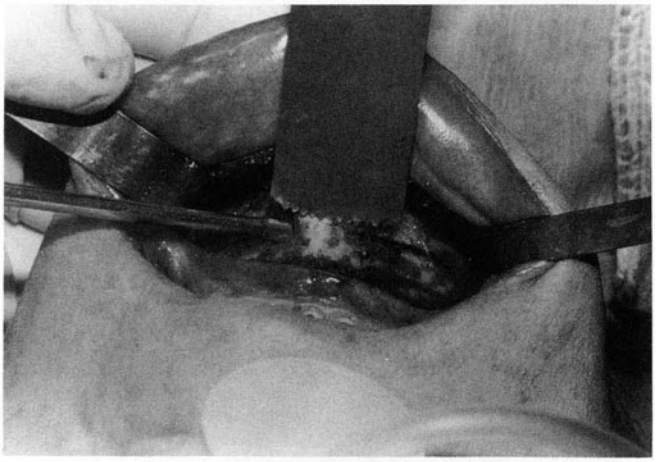
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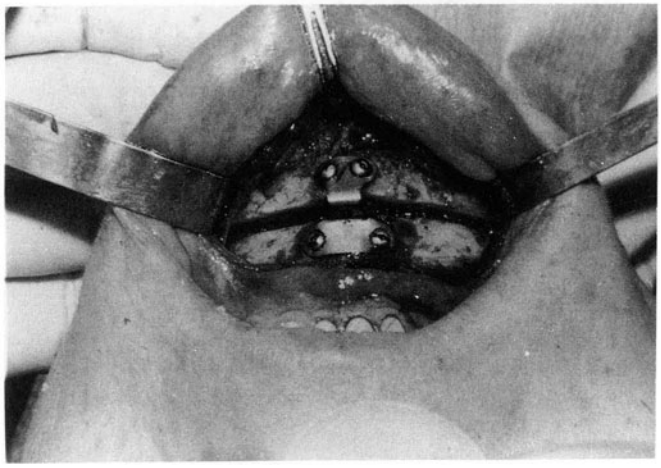
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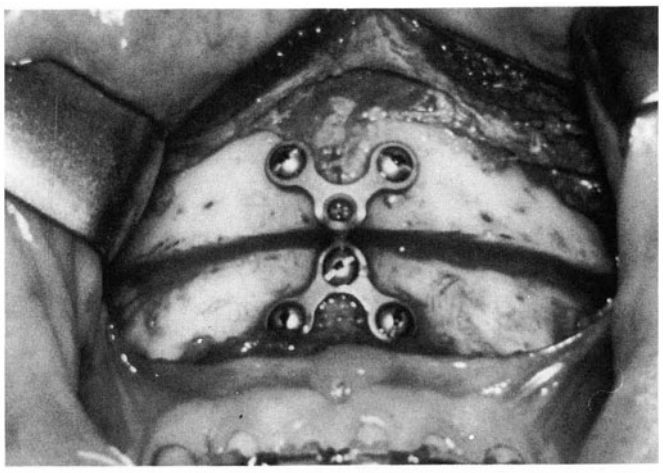
Figure 15.12. (A) The incision is made on the labial side of the sulcus, leaving at least 8 mm of soft tissue cuff on the gingiva side. (B) The soft tissue is dissected down to the periosteum; the periosteum is elevated and retracted; and the chin midline and osteotomy are marked. (C) The osteotomy is started with a wide saw blade, then completed with a fine saw blade. (D) The bone is more accurately positioned with the aid of midline markings. (E) The segment is fixed with a plate with a stepwise bend.

Osteotomy

This technique provides the freedom of moving the caudal segment of the bone in every direction that is desired. Depending on the deformity, as was described earlier, the type of osteotomy is going to be different. However, the initial approach to all osteotomies is similar. Usually, this procedure is done under general anesthesia; however, the procedure can be performed under local anesthesia with a nerve block.[36] The oral cavity is again irrigated with antibiotic solution, then infiltrated with lidocaine hydrochloride containing 1:200 000 epinephrine. The incision is again similar to the intraoral augmentation genioplasty and the ostectomy. It is essential to leave a cuff of soft tissue on the gingival side for a secure repair to avoid dehiscence or root exposure due to the gingiva being retracted by the sutures (Fig. 15.12A). The periosteum is elevated and the anterior symphysis is exposed. Following adequate exposure, the midline of the symphysis is marked vertically. Next,



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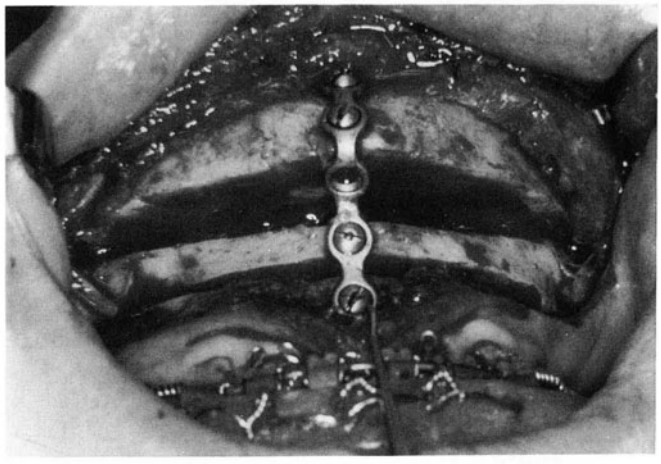

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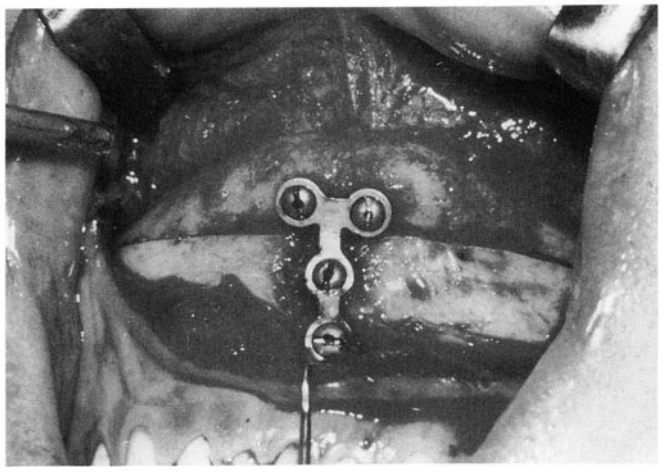


C)

Figure 15.13. (A) An X-plate was used to fix this osteotomy. (B) A straight plate has been utilized subsequent to this osteotomy. (C) An inverted T-plate is another fixation device.

the osteotomy is marked horizontally (Fig. 15.12B). If a reduction of the anterior facial height is planned, two osteotomies are marked proportional to the amount of reduction. It is imperative to perform the first osteotomy on the caudal (inferior) segment. Should the cephalad osteotomy be completed first, it will be difficult, if not impossible, to complete the caudal osteotomy. Subsequent to the osteotomies, the caudal segment is moved into a desired position and rigidly fixed (Fig. 15.12C, D, and E).

Depending on the goals, the type of fixation is going to be different. If only a setback is intended, the caudal segment is moved to the planned position and is fixed to the rest of the mandible using two screws. If a vertical reduction is planned, then a horizontal segment is resected and the caudal segment is fixed to the cephalic portion rigidly with two screws. However, should an advancement be necessary, the advanced segment is fixed in position using plates and screws.

There are a whole host of plates available that can all serve the purpose (Fig. 15.13A–C). These include step plates that would fix the caudal segment in a preplanned position based on the size of the plate. These are available in sizes ranging from 4 to 10 mm. However, one can use any type of plate and bend it to the necessary shape.

It is important to avoid any type of step deformities along the lateral boundaries of the osteotomies. If an irregularity is encountered due to excess bone, it can be corrected by removing the bone using a guarded burr. Should there be a step deformity related to a bone deficiency, it can be corrected by adding a piece of hydroxyapatite or a segment of bone.

Should there be an asymmetric chin, depending on the type of deformity, one has to decide whether the segment should be removed from one side and added to the other to maintain the anterior facial height, removed entirely to reduce the anterior facial height, or even whether a segment of bone or hydroxyapatite should be added to lengthen the lower face. Following proper alignment of the segments and rigid fixation, the wound is irrigated copiously and the mentalis muscle is repaired using 4-0 chromic catgut or 4-0 polyglactin 910, and the mucosa is repaired with 4-0 chromic catgut sutures. Finally, a pressure dressing is applied. These patients receive antibiotics perioperatively and 5 days postoperatively using a first-generation cephalosporin.

Important precautions necessary for a successful osteotomy are as follows:

1. The osteotomy should be designed at least 5 mm below the roots of the longest teeth, which are the canines. These are usually 25.5 mm long, requiring placement of the osteotomy about 30 mm from the incisal edge.
2. It is important to remember that the mental foramen extends caudally prior to its emergence through the mandibular bone and, therefore, planning the osteotomy too close to the mental foramen in the horizontal plane might result in transection of the mental nerve.
3. The retractors that are used to provide exposure will have to be pulled gently to avoid avulsion of the mental nerve.
4. Advancement genioplasty is not the correct choice for a patient with pseudomicrogenia. These patients are best served with a maxillary osteotomy.

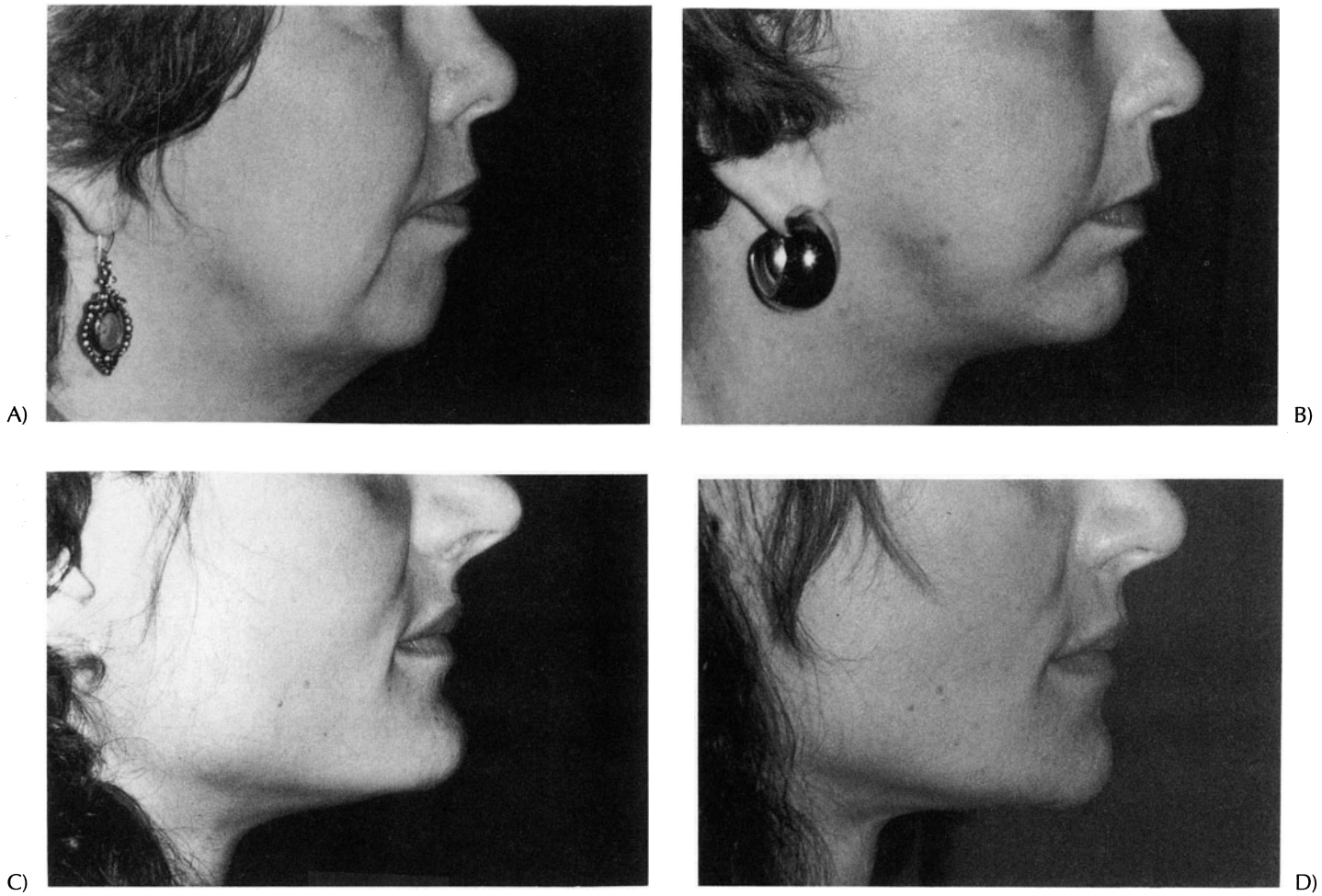


FIGURE 15.14. (A) Preoperative view of a patient who is a candidate for genioplasty and correction of aging face (A) and subsequent to rhytidectomy, submental lipectomy and genioplasty using a Proplast implant (B). Preoperative photograph of a patient with horizontal macrogenia and nasal deformity (C) and subsequent to ostectomy and rhinoplasty (D).

Advantages of Osteotomy

A lesser amount of foreign material compared to augmentation genioplasty is one of the advantages of this procedure. This operation is very predictable and one can achieve desirable results consistently. The procedure is beneficial to the neck contour and improves the cervicomental relationship. Also, theoretically, there is less chance of infection and better patient acceptance. However, the most important advantage of this procedure is its versatility, which facilitates three-dimensional movement of the caudal segment, as compared to augmentation genioplasty, which only affords alteration in an anterior direction and rarely in a caudal direction.

Disadvantages of Osseous Genioplasty

A common need for general anesthesia is a disadvantage of this procedure. Although this procedure can be performed under local anesthesia with intravenous sedation, general anesthesia is preferred.

Because this procedure is more complex, it requires more training than augmentation genioplasty to reduce the chance of intraoperative complications. Supposedly, there is a slightly higher chance of paresthesia and discomfort, but this has not been substantiated by recent studies.[37] This operation is somewhat more time-consuming and, further, it requires power tools, which are not part of the usual collection of office surgical equipment.

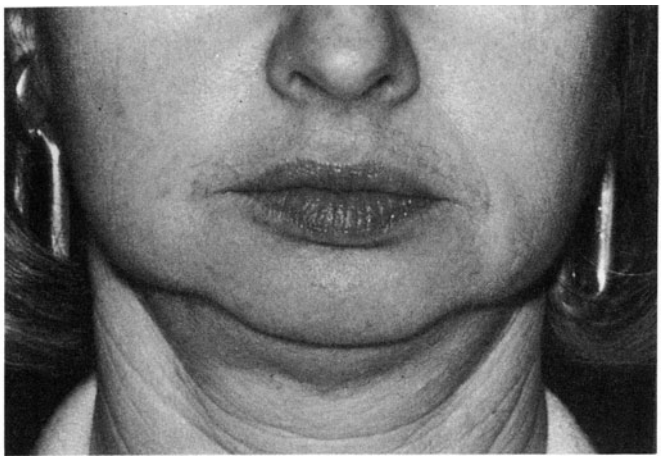
Complications

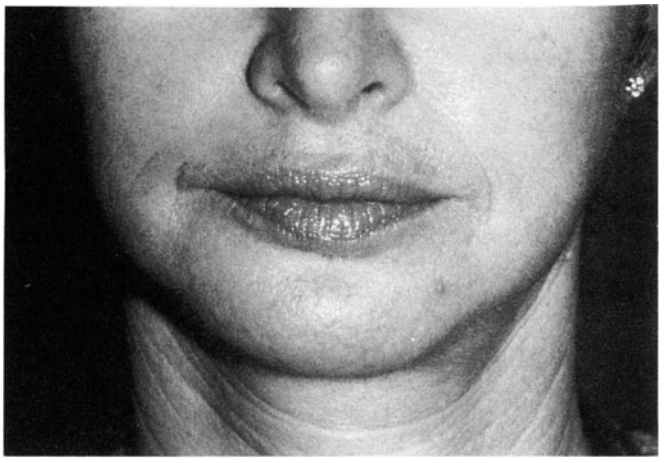
The complications related to osseous genioplasty include wound dehiscence, which is extremely unlikely and can even be minimized if an adequate amount of soft tissue is left on the gingival side of the incisive edge. Infection has not been observed in our practice, although this is a possibility with any surgical procedure. Hematomas are very unlikely; however, if discovered, these should be drained to reduce the recovery period and avoid muscle function limitations resultant from the formation of scar tissue. Permanent paresthesia is an

E)

F)

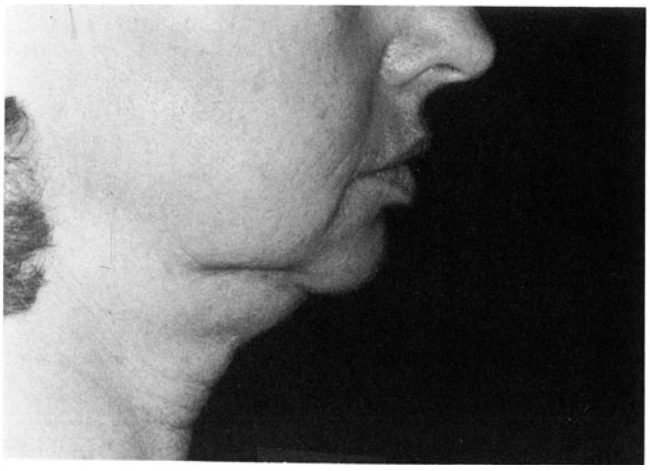
G)



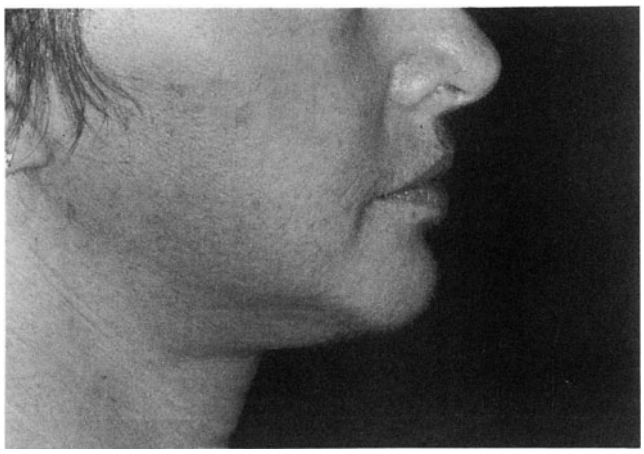


H)

FIGURE 15.14. *Continued.* Preoperative photograph of a patient with significant horizontal microgenia (E) and subsequent to advancement genioplasty (F). Front view photographs of a patient with combination horizontal and vertical microgenia (G) and subsequent to a horizontal osteotomy and anterior and caudal advancement with bone graft (H).



I)



J)

K)

L)

Figure 15.14. *Continued.* Profile views of the same patient preoperatively (I) and postoperatively (J). A profile view of a patient with combined vertical excess and horizontal deficiency (K) and following surgery (L).

unlikely possibility. Chin soft tissue ptosis can occur, especially if the chin is set back.

Root exposure due to gingival retraction is a function of poor incision placement, although infection in the gingiva may also retract the soft tissue. Usually, asymmetry does not occur if strict adherence to technical details is practiced. It is for this reason that the author marks the midlines first before an osteotomy is performed. Because complete correction of an asymmetric chin to a perfectly symmetrical one is not an easy task, one should aim for a perfect result, but not be too disappointed if it is not obtained. The patient should also be aware of the possibility that the result may be less than ideal.

It is also critical to advance the chin conservatively on patients with long face deformity who are not undergoing vertical reduction of the maxilla. These patients already have a long face and advancing the chin will make their faces look longer (Fig. 15.14A–F).

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CHAPTER 16

Mandibular Excess and Deficiency

James W. Ferraro

Until the early 1970s, the correction of all dentofacial deformities was concentrated in the dentition. Orthodontists were left with the burden of correcting the disproportion of dental and jaw relationship by dental manipulations alone, with surgery being restricted to the most severely deformed patients. The problem was that the orthodontist is very limited in the amount of discrepancy he or she can overcome and many of these cases were overtreated and, as a result, relapse and poor final aesthetic results were frequent. This situation changed with the improvement of surgical techniques, and now the maxillofacial surgeon working in concert with the orthodontist can offer the patient the best results with a minimum of relapse.

The current usual treatment plan is orthodontic manipulation of the teeth preoperatively, anywhere from 15–24 months, followed by surgical intervention and finishing orthodontics of 3–9 months. In this way the dental compensations so frequently seen in untreated Class II or III malocclusions are overcome and the dentition can be placed over basal bone. This may result in a temporary worsening of the occlusion but in this "anticipatory orthodontic" treatment plan the jaw and dentition are positioned so that when the surgery is done, the best possible relationship can be established so that relapse will not be as much a factor and postsurgical orthodontic treatment can be kept to a minimum.

The timing of surgery is usually dependent on the completion of growth. In his book, Bell,[1] using Scammon's[2] curve, found that facial development falls between the noural curve and the general body curve. The growth curve of the brain follows the neural curve and the cranial base falls very close to this growth curve also. The maxilla, being slightly caudad to the brain, is more influenced by both the neural and general body curve. The mandible is even farther from the brain and, as a result, follows the general body curve even more closely (see Fig. 16.1) As a result we see the completion of growth of the maxilla at around 15 years of age, whereas the mandible continues to grow until around 16–19 years of age, with growth in females completed before males.

The history of mandibular osteotomies to correct jaw deformities began with Hullihen[3] in 1848, when he resected a portion of the anterior body of the mandible to correct a burn scar contracture deformity of the mandible. Subsequently, work by Blair,[4,5] New,[6] and Dingman[7] continued to correct malocclusions associated with the mandible by resecting a portion of the body of the mandible. Later, condylectomy was proposed by Gonzalez-Ulloa[8] and Merville,[9] and sigmoid ostectomy by Smith and Robinson.[10] All these osteotomies are, for the most part, of historic interest only and are not used today. Figure 16.2 shows these historic osteotomies.

By 1954, the work of Caldwell and Letterman[11] described an extraoral approach to the ramus of the mandible to perform a vertical subcondylar osteotomy. This was used most successfully for mandibular setbacks, with the interaoral version of this procedure frequently being used today. This type of osteotomy will be described later in this chapter.

The lengthening of the mandible is best served by the saggital split osteotomy, which gives a good overlapping bone to bone contact as the mandible is moved anteriorly. This was originally described by Trauner and Obwegeser[12] and modified by Dal Pont,[13] Hunsuck,[14] and Epker.[15] This procedure is also widely used for decreasing the projection of the mandible in Class III malocclusions. The one drawback to this procedure is the frequent postoperative anesthesia associated with a sagittal cut in the ramus region.

In his article on the long-term follow-up evaluation of patients with sagittal split osteotomies, Pepersack[16] reported a 60 percent incidence of some impairment of

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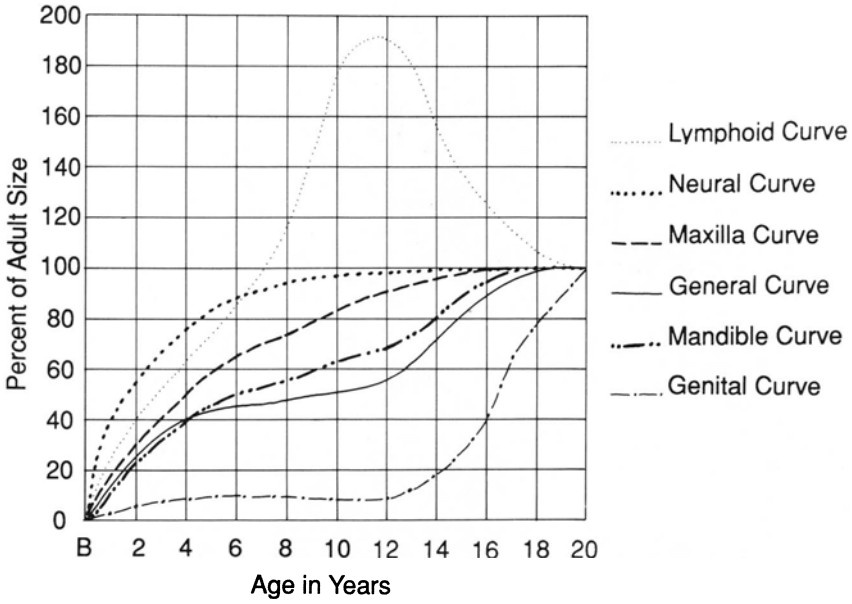

Figure 16.1. Growth curves of lymphoid tissue, neural tissue, the maxilla, and mandible from birth to adulthood.

sensation in the lower lip. Freihofer[17] also reported an incidence of 72 percent of decreased sensation to the lower lip. Pepersack however, did state that 40 percent of his cases went unnoticed and were detectable only with direct testing of the lip sensation.

Mandibular Excess (Prognathism—Class III)

In patients with mandibular excess the lower third of the face is very prominent. However, increasingly we are noting that some of these patients present with a prominent lower third but in reality the maxilla is really deficient. It is my strong feeling that when the maxilla is flat and the canine fossa is concave, even if the mandible may be slightly excessive, that surgical correction should be confined to the maxilla so as to give the best aesthetic result.

Physical Evaluation of the Prognathic Patient

The facial appearance of the prognathic patient in the frontal view exhibits:

1. A broad lower third of the face.
2. Reduction of the labiomental fold with a full lower lip.
3. The lower vermilion is thin.
4. Alar bases may be normal or narrow.

The evaluation of the profiles of these patients is related to the lips and tongue and their effects on the dentition, which result in dental compensations:

1. Because there is an anterior crossbite, the muscles of facial expression, especially the orbicularis oris muscle, produce a force on the lower incisors. This results in a tilt of these lower incisors lingually.
2. The tongue has increased space because of excessive jaw size and the forward jaw position; therefore it lies in the floor of the mouth and has less influence on the palate and the maxillary arch. Thus, the maxillary arch is frequently constricted and the maxillary central incisors are more lingually inclined, producing less support to the upper lip. As a result, the lip is frequently rolled in, which results in less vermilion show in both profile and frontal views.
3. The prominent lower teeth cause a rolled out appearance to the lower lip, with a fullness to the lower lip just below the lower vermilion border and a decrease in the labial mental sulcus (Fig. 16.3).

When Class III malocclusion is associated with overclosure, there is a decrease in vertical facial height and this decrease can accentuate the prominence of the lower third of the face. In the evaluation of such cases where you are considering a change in vertical dimension, simply have the patient relax their jaws to increase their interincisal space, which opens the bite and gives you a simple method of seeing the changes associated with an increase in vertical dimension (Fig. 16.4).

Surgically, the correction is done either by a down fracture and inferior placement of the maxilla or by correcting the overclosure of the Class III malocclusion and establishing a Class I dental relationship. Figure 16.5 shows the corrected occlusion, which also resulted in an increase in vertical dimension.

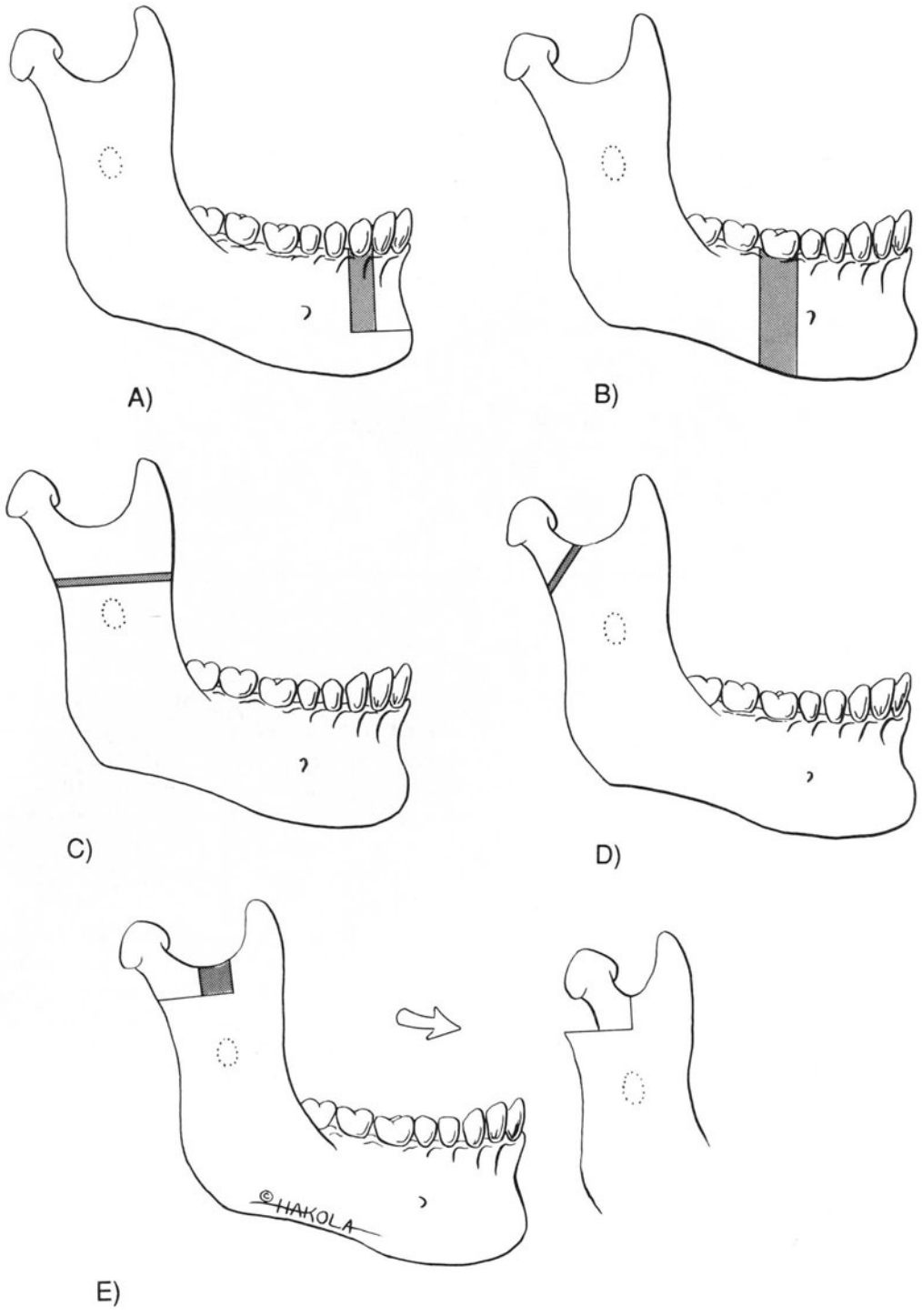

Figure 16.2. Historic osteotomies of the mandibular (these are rarely used today). (A) Similar to Hullihen; (B) Body ostectomy; (C) Horizontal osteotomy; (D) Subcondylar osteotomy; (E) Smith and Robinson condylar osteotomy and setback.

Figure 16.3. Lower lip fullness and prominence below vermilion border as seen frequently in patients with Class III maloclusion.

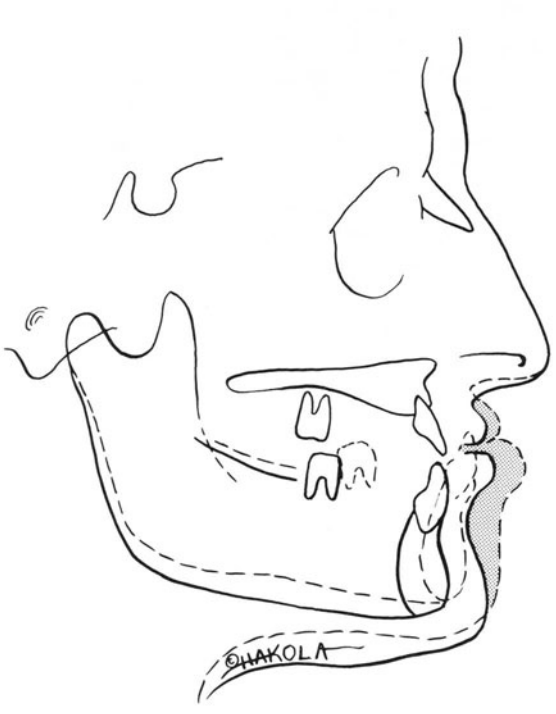

Figure 16.4. Changes in profile with changes in vertical dimension.

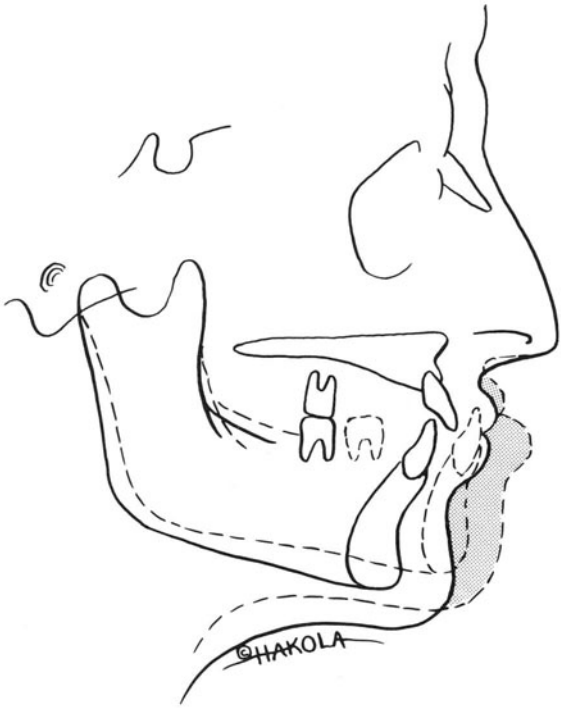

Figure 16.5. Correction of overclosure by establishing a Class I occlusion.

Cephalometric Evaluation of the True Prognathic Patient

1. Minimal distance from articulare to gnathion
2. Increased SNB angle
3. Normal SNA and Landes Angle
4. A negative angle of convexity
5. Obtuse facial plane angle
6. Normal S-N-O angle
7. FMA is usually normal or more acute
8. Lower facial throat angle of approximately 100–117 degrees.

Cephalometric Evaluation of the Pseudoprognathic Patient, or a Patient Who Would Need a Maxillary Procedure Even Though the Profile Is Concave

1. SNB angle normal or slightly increased.
2. SNA decreased.
3. Negative angle of convexity.
4. Slightly obtuse or normal facial plane angle.
5. S-N-Or angle normal or acute.
6. Lower facial throat angle increased.
7. Ar–Pg may be normal or slightly enlarged.
8. Lower facial height/lower facial depth is greater than one.

Surgical Procedures to Correct Prognathism

Transoral Vertical Ramus Osteotomy

The vertical osteotomy of the mandible performed intraorally (previously it was done extraorally) is an excellent method for treating mandibular prognathism. With the development of new instrumentation, the intraoral procedure has become a valuable adjunct to the maxillofacial surgeon. Its advantage to the older extraoral approach is the absence of external scar and the avoidance of external dissection, which could injure the marginal mandibular branch of the facial nerve.

The initial incision is made in the buccal sulcus quite lateral, so as to give a good soft tissue pedicle so that closure can be accomplished at the completion of the procedure when the jaws are wired together. The incision is started at the occlusal surface of the lower teeth and carried down from there. This minimizes the chances of cutting the long buccal nerve and exposing the buccal fat pad, which then becomes a nuisance throughout the rest of the procedure.

On the buccal side the massiter is stripped off the ramus on its lateral surface but not off the posterior surface (Fig. 16.6). Right and left Bauer retractors are placed, one in the sigmoid notch and one at the inferior border of the mandible (Fig. 16.7). This gives good exposure to the lateral ramus surface. Another good retractor, but one that requires some posterior stripping to position it, is the L-M retractor (Levassuer-Merrill), as described by Moose.[18] This retractor hooks over the posterior surface of the posterior border of the ramus and affords excellent visualization.

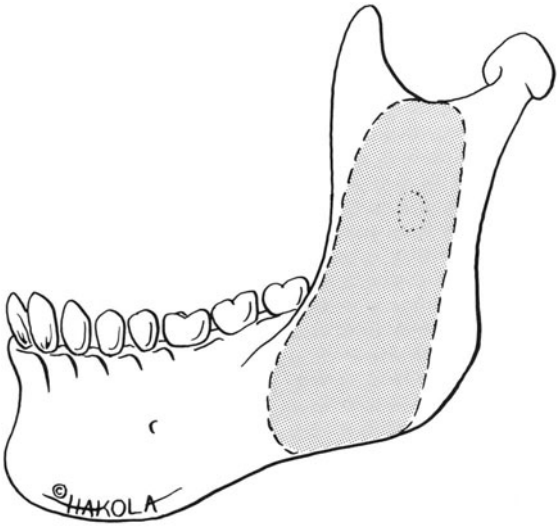

Figure 16.6. Area of stripping in vertical osteotomies.

The osteotomy is done with an oscillating saw blade, angled about 30 degrees from the shaft, so that a beveled cut is made. This does two things: (1) it has the cut going away from the alveolar canal and (2) it affords a bevel so that more bone to bone contact can occur once the overlapped segments come into contact with each other (Fig. 16.8).

The osteotomy is started at the sigmoid notch and proceeds in a slight arc inferiorly. A through and through cut is made superiorly and inferiorly, leaving the completed cut to be made last in the midportion of the mandible. In this way, if the alveolar artery were accidentally injured, the osteotomy cut could be completed and the bleeding quickly controlled. It is important to note the usual bulge on the lateral surface of the mandible, called the antilingualar prominence. This demarks the inferior alveolar foramen on the lingual side. The osteotomy cut should be slightly posterior to this area.

Once the osteotomy is completed the condylar fragment tends to be distracted medially. It can be grasped and the internal pterygoid muscle attachment stripped off just enough to give bone to bone contact. The condylar fragment must be positioned buccally to the distal (dental) segment. The internal and external pterygoid muscles will pull medially and hold the proper relationships where the mandible has been retruded into its proper occlusion.

After the osteotomy cuts are completed, the corrected occlusion is established. The overlapped segments are

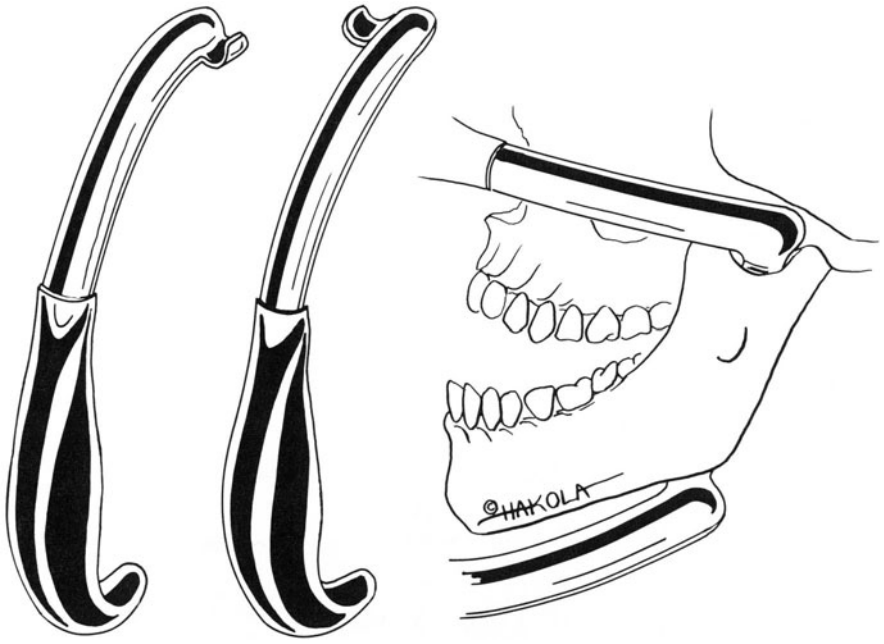

FIGURE 16.7. Right and left Bauer retractors: one placed at sigmoid notch and one at the inferior border to give good exposure to the lateral ramus surface.

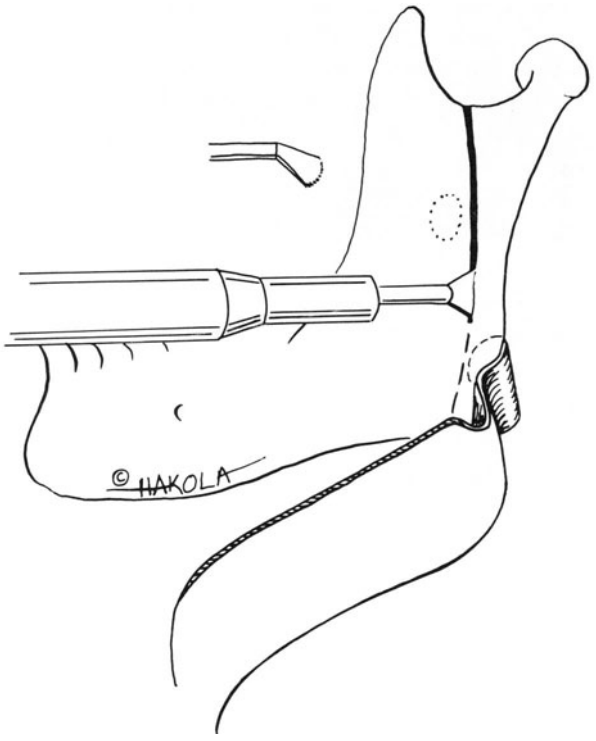

FIGURE 16.8. Thirty-degree oscillating saw making the vertical osteotomy cut in the mandibular ramus, with Levassier–Merrill retractor in place.

positioned passively against each other and the wounds are closed in two layers with an absorbable suture material.

There are several additional points that should be mentioned here:

1. Hayward[19] has mentioned that measurements have shown that the mandibular groove (which is just above the inferior alveolar foramen) is 5–7 mm from the posterior border in most patients. Because the vessels are coming into the foramen at a superior medial angle, an angular saw blade that is 5–7 mm long wll be cutting away from these vessels and be safe. One advantage of using the L-M retractor is that its posterior curve is 5 mm from the hooked end, which helps guide us in this vertical cut.

2. Usually osseous fixation is not required in patients undergoing vertical osteotomy of the mandible. In most of these cases simply letting the segments lie passively against each other will permit osseous union. Some surgeons, however, feel that to prevent postoperative condylar sag and subsequent malocclusion, the osteotomized segments must be wired together (Fig. 16.9). However, all these patients do require maxillomandibular fixation for a period of 4–6 weeks. Frequent evaluation is needed after the intermaxillary fixation is removed. The use of elastic traction is needed to be cer-

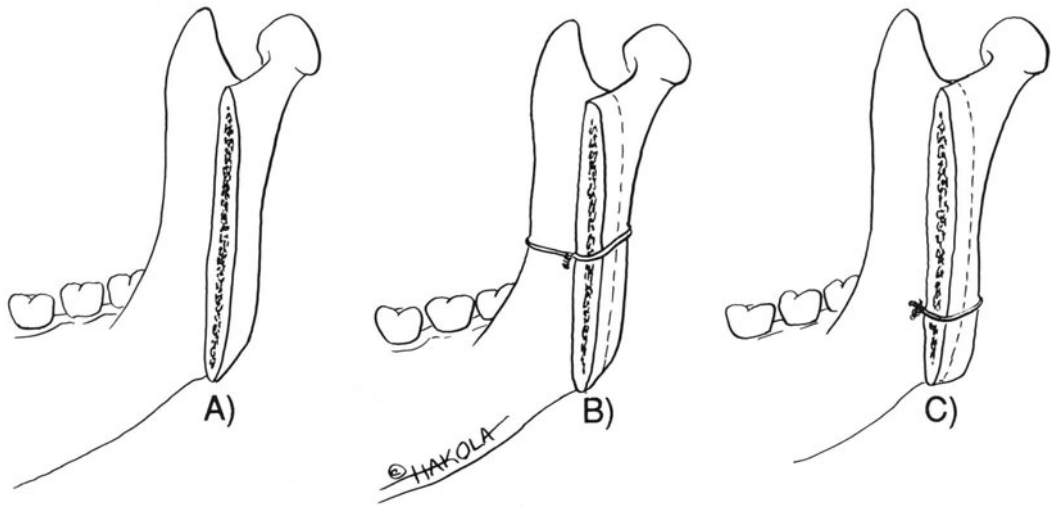

Figure 16.9. Overlapped vertically osteomized segments. (A) Without fixation. (B,C) with wire fixation.

tain the occlusion doesn't open. Instability at the osteotomy site can cause condylar sag and a development of shift in the occlusion. However, the patient is aware of these changes when instructed and can come in for adjustment of their elastic traction.

3. This procedure should not be used to advance the mandible, as in Class II malocclusions, or to close an anterior open bite.

4. If the overlapped segments do not lie flat against the distal segment (dental segment) then a burr should be used to smooth off the points of premature contact in the medial aspect of the condylar segments, so that good bone to bone contact is established.

5. In extreme mandibular prognathism an inverted L-osteotomy may be an alternative procedure that can be used instead of the standard vertical osteotomy, in conjunction with a coronoidectomy (Fig. 16.10).

Mandibular Deficiency (Retrognathia—Class II)

In data from the U.S. Public Health Survey, 2.5 percent of youth in the United States from 12–17 years of age have mandibular excess, with an end to end dental relationship or an anterior crossbite. This is in comparison to 20 percent for youths aged 12–17 who have mandibular deficiency. There are, however, many causes of mandibular deficiency, such as intrauterine molding (Pierre–Robin Syndrome), condylar agenesis, Treacher Collins, hemifacial microsomia, trauma in childhood to the mandible, ankylosis secondary to trauma or infection, juvenile rheumatoid arthritis, and lastly the idiopathic type, which may be an inherited or unknown cause of a developmentally small mandible. Another group to consider are those who appear to have a mandibular deficiency, but have a long face syndrome with counterclockwise rotation to the mandible.

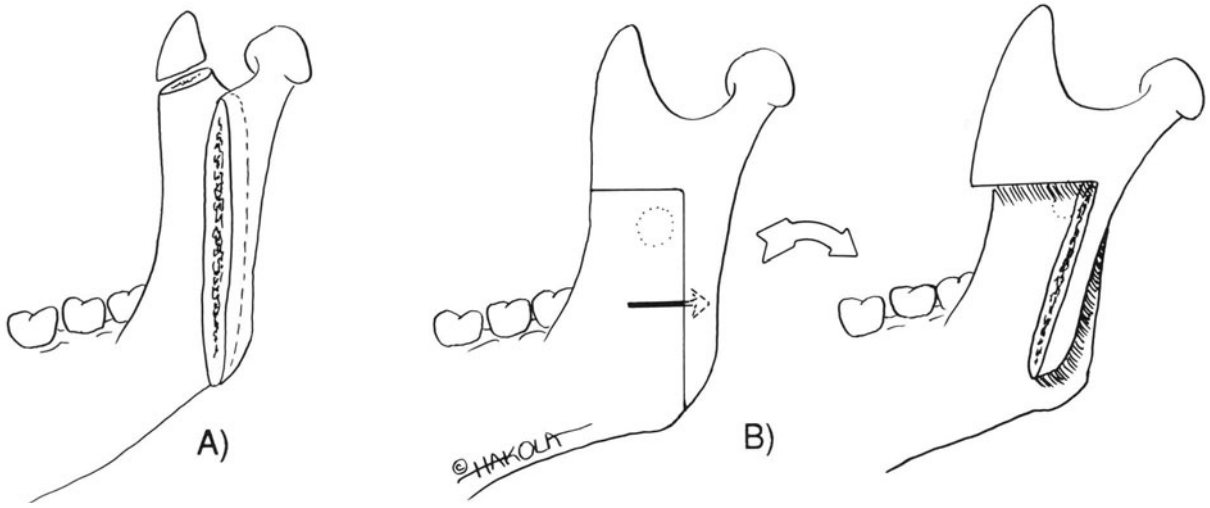

Figure 16.10. (A) Vertical osteotomy with coronodectomy. (B) Inverted "L" osteotomy.

Physical Evaluation of the Retrognathic Patient

The facial appearance of the retrognathic patient in the frontal view exhibits:

1. Depending on the severity, the lower third of the face may be: (1) normal; (2) deficient, if there is retruded mandible and/or closed bite; or (3) long, if there is a long face or open bite with the mandible rotated inferiorly or posteriorly.
2. Protruding anterior teeth with its associated lip incompetence. The lower lip frequently is curled up under the maxillary central incisors.

The evaluation of the patient in the profile view:

1. In Class II Division I, the maxillary arch is frequently v-shaped instead of round, with the narrowing occurring in the cuspid to bicuspid region. There is flaring of the anterior teeth with resultant lip incompetence. This results in a mentalis strain pattern that frequently results, over time, in the flattening of bony pogonion, so that its anterior to posterior measurement is closely related to Pt. B (the dental base) (see Fig. 16.11A).
2. Because the mandible is retrognathic, the tongue position in the palate is altered. As a result these patients have a high incidence of tongue thrusting with swallowing. There is excessive buccinator activity in an attempt to get lip competence with swallowing and this, in part, is why the arch may be narrow in the bicuspid region.
3. Class II, Division II—Here, the maxillary arch is seldom narrow as in Division I cases. Instead it is wider than normal and there is excessive lingual tilt to the anterior central and lateral incisors. Sometimes the central incisor is tilted lingually with the lateral incisor being flared labially. There is a deep bite with the lower incisors impinging on the palate mucosa.
4. The labiomental sulcus is deep (greater than 4 mm) and the lower lip is everted because it comes in contact with the upper incisors.
5. The lower lip vermilion is everted.
6. Depending on the amount of soft tissue pad and the severity of the retrognathia, the chin may be in normal balance or significantly retropositioned.
7. Class II molar and canine relationship.
8. Excessive curve of Spee in the mandibular arch with a mirror reverse curve in the maxilla.
9. The lower face projection appears to be decreased in profile when the Class II malocclusion is associated with an open bite or excessive vertical dimension in the maxilla (long face) (Fig. 16.11).

Cephalometric Evaluation of the Retrognathic Patient

1. S-N-B angle is decreased.
2. Angle of convexity is increased and may exceed +20 degrees.
3. Acute facial plane angle.
4. SNA and Landes angles are usually normal.
5. If there is a true short mandibular body, the Ar-Gn will be smaller than normal.
6. If the retrognathia is 2 degrees to long face, the FMA will be obtuse and the upper facial height to lower facial height will be greater than 1:1.
7. The lower facial throat angle is obtuse (120 degrees or more).
8. Obtuse facial contour angle.

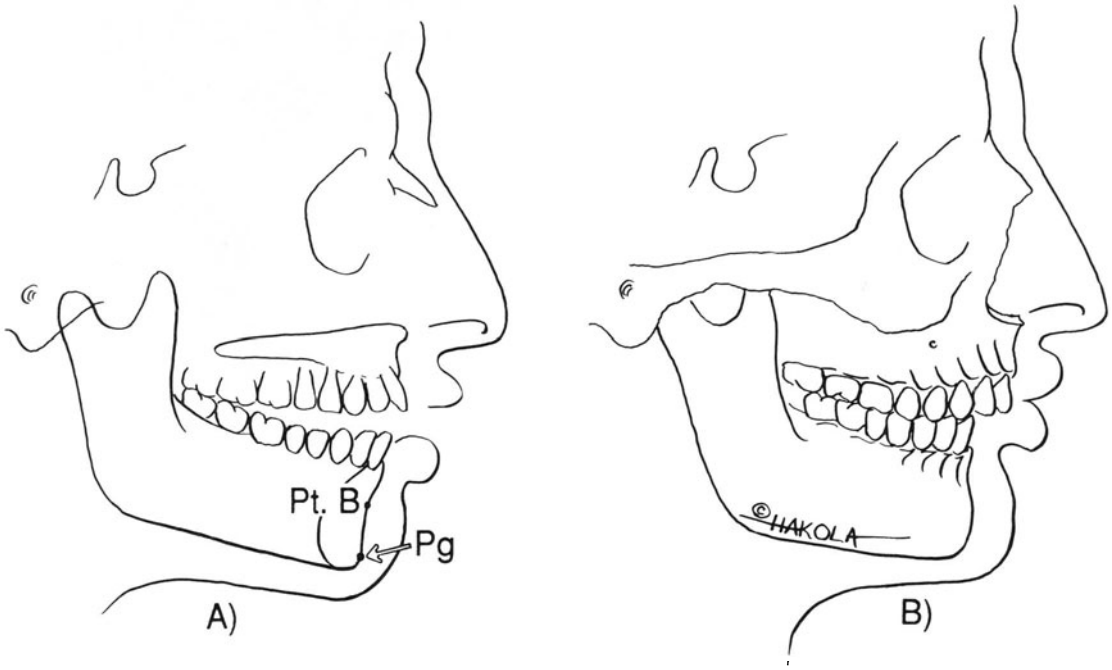

FIGURE 16.11. (A) The typical open bite patient with Pt. B being almost equal in A-P dimension to Pg and the somewhat flat chin indicating the mentalis strain. (B) The deep labiomental crease and the everted redundant lower lip.

Surgical Procedure to Correct Mandibular Deficiency

Bilateral Sagittal Split Ramus Osteotomy

The sagittal split ramus osteotomy is the preferred procedure for Class II malocclusions where mandibular advancement is desired. As mentioned earlier, it also will adapt for use in patients with mandibular excess where the mandible is to be retruded. The procedure has been enhanced by new instrumentation. Now the only major concern is to stay away from the inferior alveolar nerve when splitting the mandible, and being sure that the condyle is correctly seated in the glenoid fossa before position screw fixation is performed.

As with the vertical osteotomy, the initial incision is made well out into the buccal sulcus so as to leave a good pedicle of soft tissue. This becomes important when closing the soft tissue. The incision is started at the level of the occlusal surfaces of the lower teeth and carried to about the second bicuspid region. Less ramus stripping on the buccal is necessary in this surgical procedure when compared to the vertical osteotomy (Fig. 16.12). Now using the Hargis anterior ramus stripper, the temporalis attachment to the anterior ramus margin is removed up toward the coronoid process. To facilitate exposure a Kocher clamp is used to grasp the coronoid to help retract the soft tissues. Now a medial tunnel is made above the inferior alveolar foramen from the anterior border to just behind the mandibular fossa (Fig. 16.12B).

An Obwegeser channel retractor is placed (Fig. 16.13), in the medial side in the tunnel that has been created. This protects the nerve, artery, and veins that come into the inferior alveolar canal. A barrel burr is used to remove a rounded slot on the anterior oblique ridge. This gives the ability to see along the medial aspect of the ramus. Now a Lindeman side-cutting burr is used to complete the medial osteotomy to a depth of one half the thickness of the ramus (Fig. 16.13A).

The osteotomy of the anterior border of the ramus is performed with an oscillating saw down to the region between the first and second molars (Fig. 16.13B). The completion of the vertical cut in the ramus is made with a reciprocating saw, making sure that the cut at the inferior border of the mandible goes around the inferior border to the lingual side (Fig. 16.13C and inset). Now using curved osteotomes with the curve toward the buccal side, the osteotomy is completed (Fig. 16.13D). Care must be taken to completely separate the osteotomized segments so that advancement of the dental segment can be made without inadvertently pulling the condylar segment out of the glenoid fossa.

The jaws are now placed in the predetermined corrected occlusion. The condylar segment is positioned by an assistant so that the condyle is seated in the glenoid fossa. Luhr[20] and others have written on the intraoperative control of the segments so that the guesswork in the position of the condyle is eliminated. Fixation plates between the ramus and the maxilla are placed before the surgical cuts and then these plates are removed, the surgical split done, and the plate replaced at the time of approximating the cut ramus to the newly positioned dental segment. This places the condyle in exactly the preoperative position, so that when the position screws are placed the condyle is not in a detracted position in the glenoid fossa.

Once the occlusion has been established and the condyle seated we are ready to fix the bony fragments

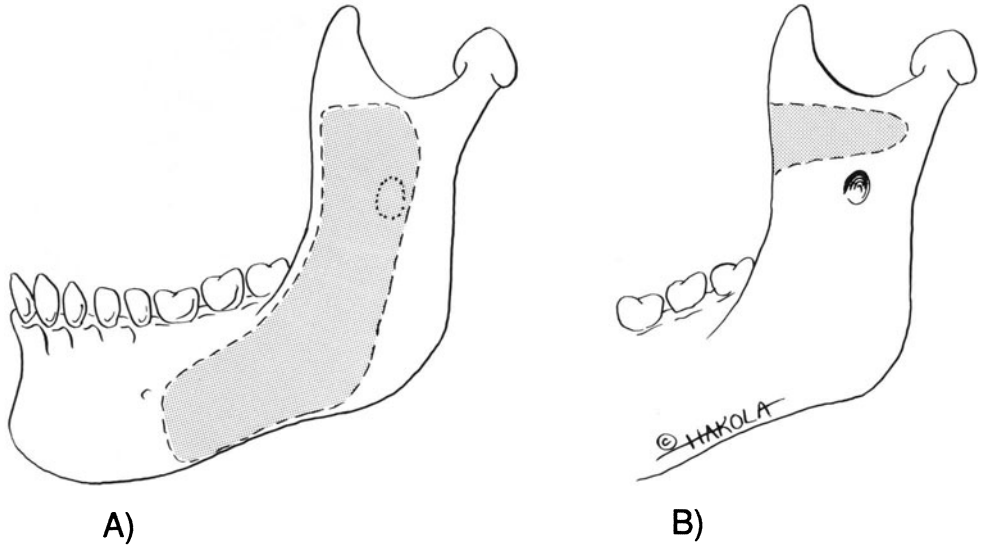

FIGURE 16.12. Amount of periosteal stripping. (A) Buccal view; (B) Lingual view.

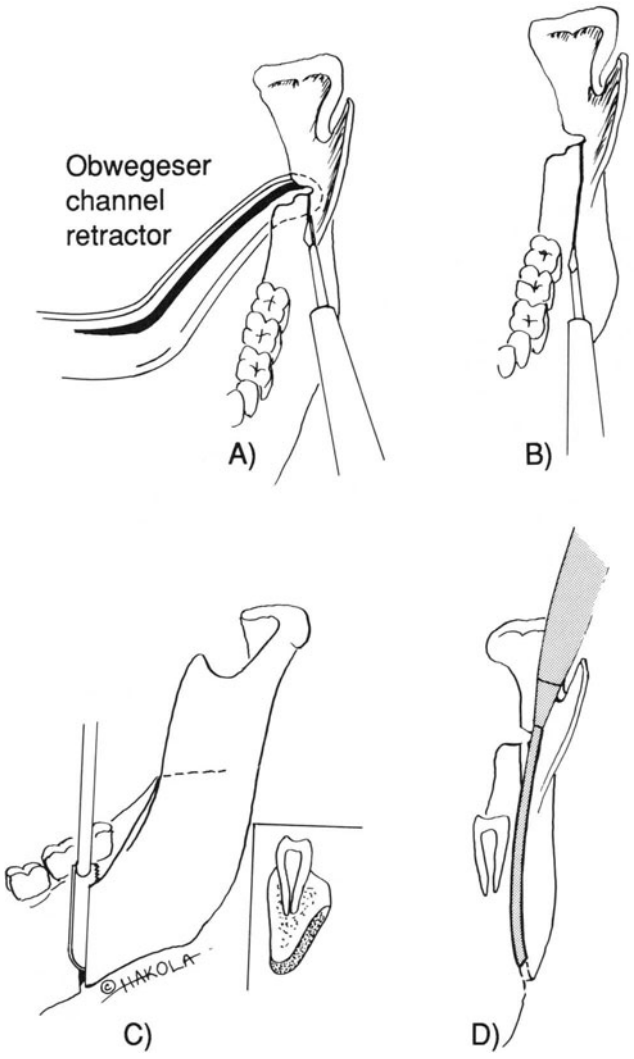

Figure 16.13. (A–D) Bilateral sagil ramus sagittal-splitting ramus osteotomy.

with screws. There is new instrumentation available with an elongated right angle screwdriver that permits the intraoral placement of the screws without the need for a trocar and external incision. However, as of this writing the most common method used is the trocar system. This requires a 2 mm stab incision in the lateral submandibular area to place the screws. The trocar is used with a shim to stabilize the turning drill bit and facilitate an accurate drill hole. The shim is then removed and a screw is passed down the shaft of the trocar into the bone. As discussed by Foley[21] and Ardary,[22] the best configuration for screw placement is the inverted L position, with two screws in the superior border and one at the inferior border (Fig. 16.14A). This configuration was stronger than when three screws were placed in a row (Fig. 16.14B).

Now that the screws have been placed, the patient is removed from intermaxillary fixation and the occlusion is evaluated. The intraoral wounds are irrigated and closed in two layers with 3-0 chromic. I prefer to keep my sagittal split patients in intermaxillary fixation for several weeks (3–4) after surgery, even though three screws are placed on each side. This is because the screws are used only to loosely approximate the bony surfaces, whereas the tightened screw could easily crush or injure the inferior alveolar nerve in its new bony relationship.

Also the advanced segment usually doesn't lie passively against the condylar segment and, as reported by Raveh,[23] if the screws were ratcheted down it could easily displace the condyle laterally in the glenoid fossae (Fig. 16.15). Many surgeons, however, do not choose to use any postoperative intermaxillary fixation after sagittal split osteotomy when three screws are placed on each side.

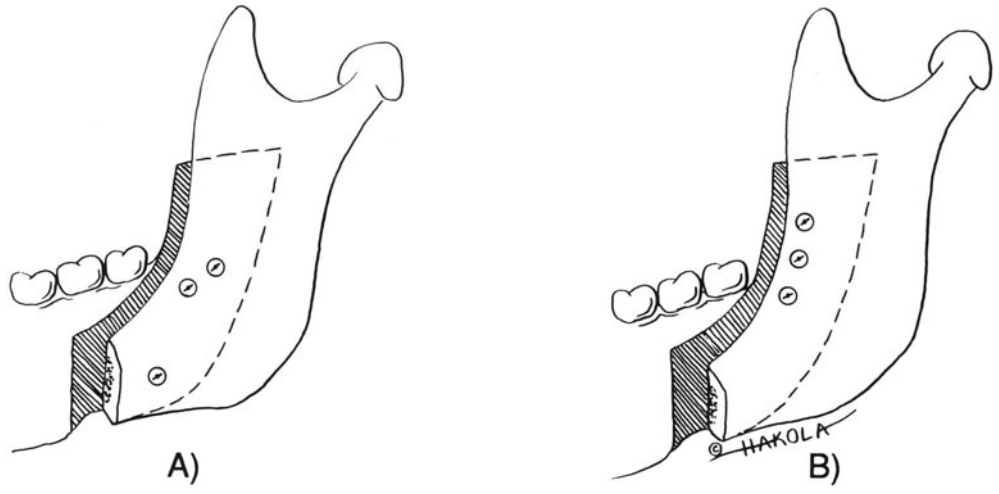

Figure 16.14. Screw placement—Inverted "L" preferred over horizontal placement.

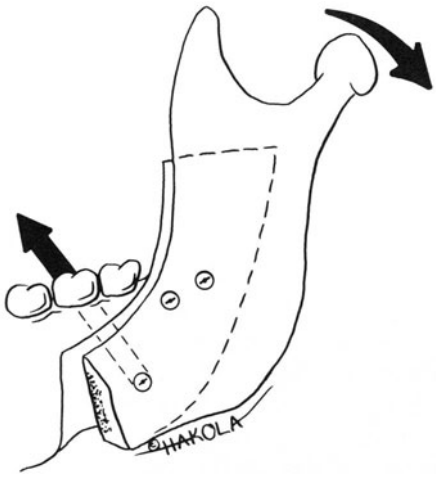

Figure 16.15. Condylar displacement secondary to firm screw placement.

Some additional points about the sagittal split osteotomy should be considered here:

1. *Inverted L- or C-osteotomy*—There are a few times when the inverted L- or C-osteotomy in the ramus can be invaluable. This is especially true in the patient with hemifacial microsomia, with a small ramus that frequently has less cancellous bone and as a result will not split along conventional lines. In these cases the intraoral or extraoral inverted L- or C-osteotomy can be used to advance the mandible and/or change its vertical height. These osteotomies can also be combined with bone grafts to fill voids created by the deformity or the osteotomized segments (Fig. 16.16).

2. *The position of the inferior alveolar nerve in the bony canal*—The vertical position of the inferior alveolar nerve in the bony canal is an important consideration in several phases of the sagittal split osteotomy procedure. During the osteotomy, in the anterior surface of the ramus just medial to the external oblique ridge, it is possible to injure the nerve with the sagittal saw. As reported by Smith,[24] the mean vertical distance to the canal was 10.9 ± 2 mm at the mandibular trigone region.

The distance from the nerve canal to the inferior border of the mandible at the distal root of the second molar, the bed of the third molar, and at the junction of the ramus with the body was 8.3 ± 1.5 mm, 8.9 ± 1.8 mm, and 12.3 ± 2.7 mm. Therefore, when making the vertical cut of the sagittal split with a reciprocating saw at the inferior border, this distance must be considered. These measurements are also important when considering the placement of screws for osteosynthesis.

3. Also in the report by Smith[24] it was found that the total thickness of the mandible and, in particular, the lingual cortical bone at the external oblique ridge, was significantly wider than at the inferior border and therefore screws placed here would be more apt to hold (Fig. 16.17).

4. The medial portion of the sagittal split osteotomy does not go back to the posterior border of the mandible. As reported by Hunsuck[25] and Mercier,[26] the osteotomy split usually occurs just behind the inferior alveolar foramen and goes vertically to the inferior border (Fig. 16.18).

5. *Complications*—As reported by Turvey,[27] the incidence of complications overall in his study was 8.2 percent of 256 patients.

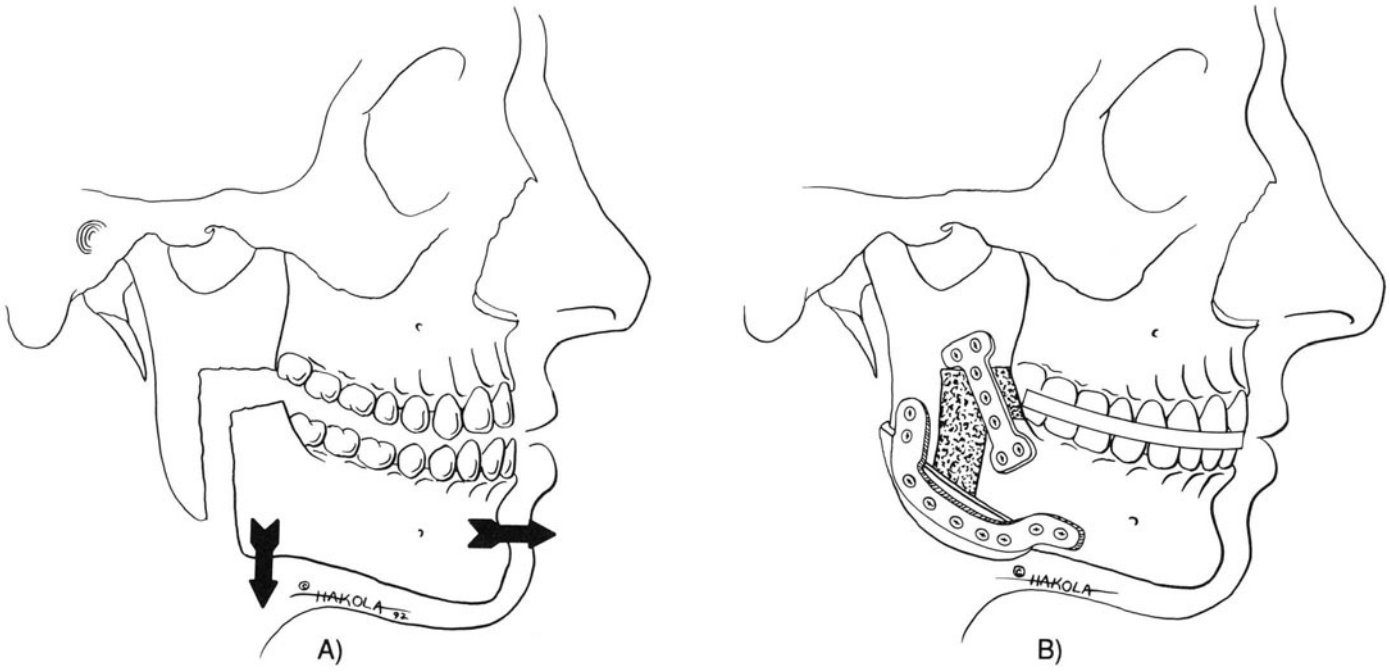

Figure 16.16. C-osteotomy with bone grafts.

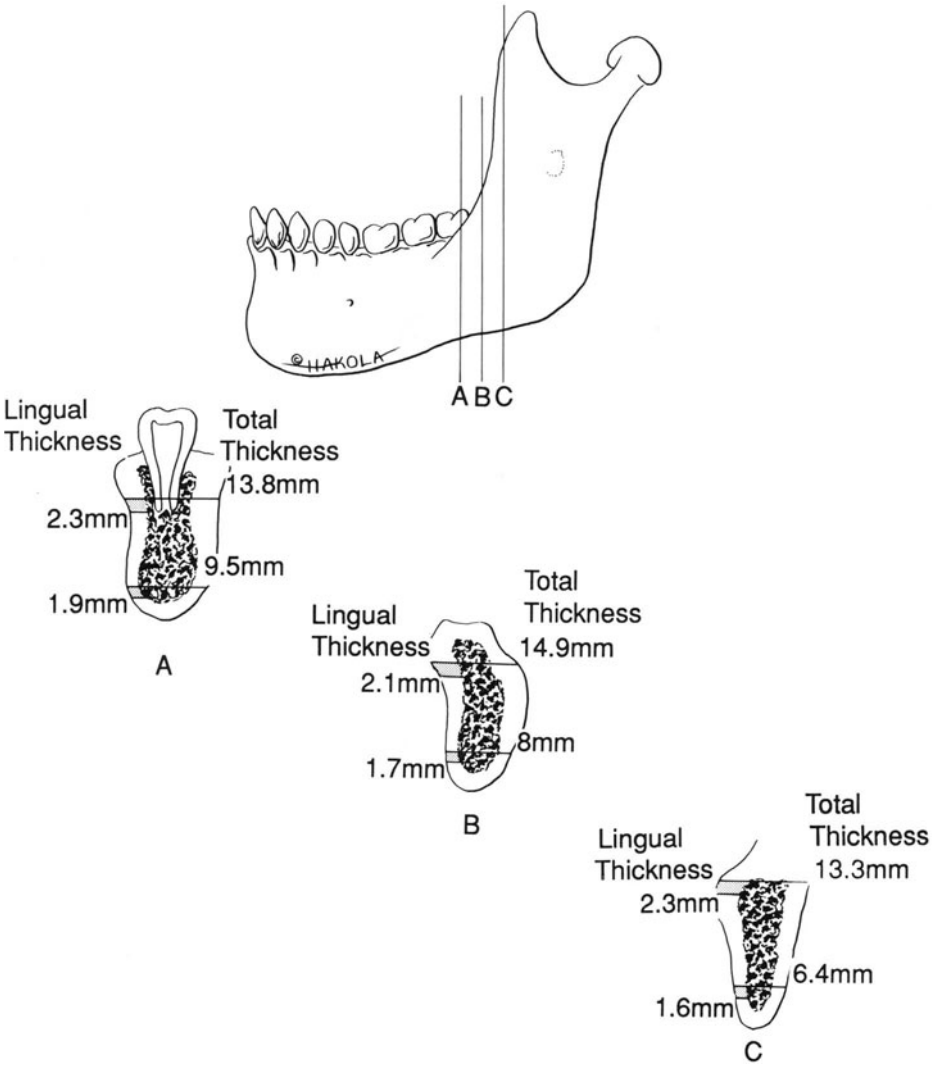

Figure 16.17. Mean thickness of mandible and mean thickness of lingual cortical plate.

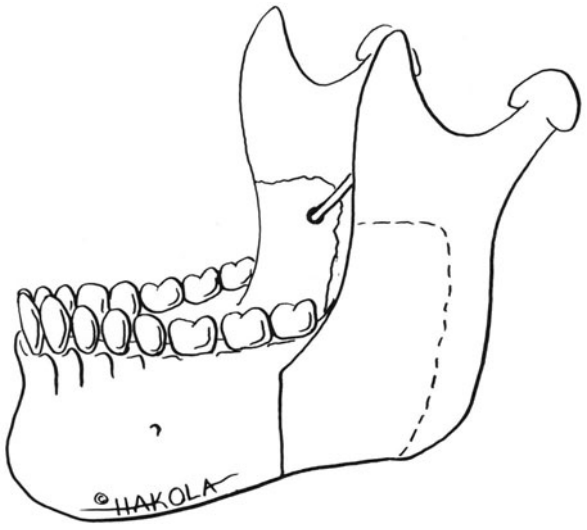

Figure 16.18. Represents the extent of the medial portion of the sagittal-split osteotomy.

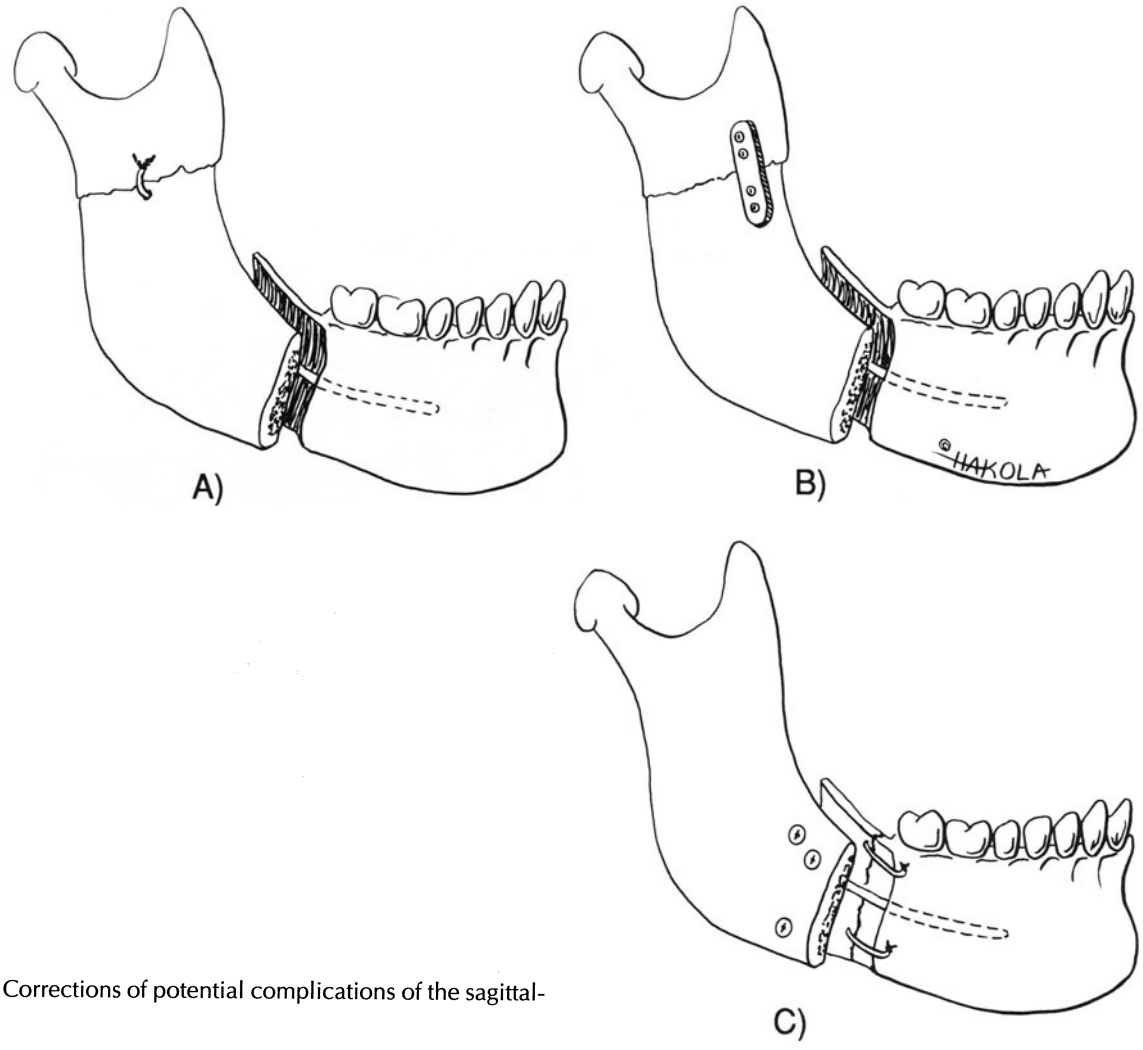

Figure 16.19. (A–C) Corrections of potential complications of the sagittal-split osteotomy.

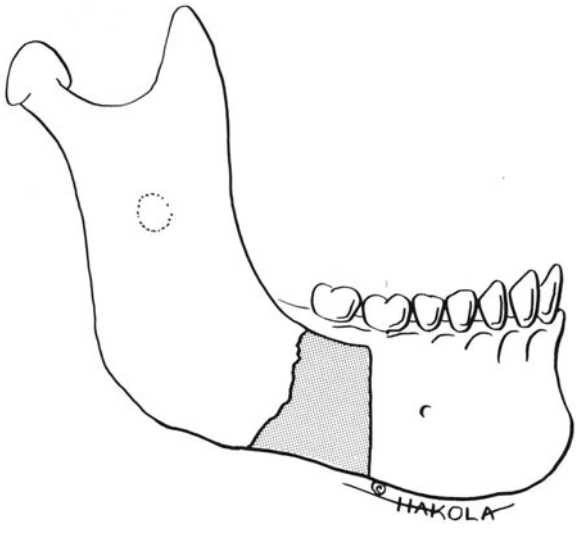

Figure 16.20. Avascular necrosis of buccal plate.

This is consistent with other authors.[28–30] Transection of the alveolar nerve occurred with the greatest frequency (3.5 percent), proximal fractures in the ramus (3.1 percent), hemorrhage (1.2 percent), and distal fractures were the least common (0.3 percent). In the proximal or condylar segment fractures, direct wiring or plating will be required so that proper positioning of the condyles in the glenoid fossa can be performed (Fig. 16.19A and 16.19B). If the fracture occurs on the dental (distal) segment it is more difficult. Careful minimal lingual dissection is necessary and a wire is placed to control the fractured segment. The mandible is advanced and the screw fixation is performed (Fig. 16.19C). In both instances, intermaxillary fixation for 6–7 weeks is indicated to permit undisturbed healing.

If the alveolar nerve is lacerated the osteotomy should be completed, the damaged nerve manipulated into position, and the screw osteosynthesis completed. As reported by Turvey,[27] no sutures were placed in these partially traumatized nerves and in his cases, in 2–5 year follow-up, all patients have had at least some return of sensation. If, however, the nerve was completely

transected, repair with 6-0 or 7-0 nonabsorbable suture is indicated.

Finally, if too much muscle stripping and periosteal elevation has been done on the buccal surface of the ramus, the blood supply to the distal (condylar) segment can be compromised. This is especially true if the Dal Pont modification of the sagittal split has been done. This could easily result in an avascular necrosis and sequestration of the extended portion of the osteotomy (that portion that was originally in the second molar region). This has also been reported by Bell and Schendel[28] (Fig. 16.20).

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CHAPTER 17

Vertical Maxillary Deformities

Stephen A. Schendel

Vertical maxillary deformities were rarely recognized or treated before the early 1970's. Their recognition is based on changing aesthetic mores and increasing sophistication of cephalometric analyses. Classical cephalometric analyses paid little attention to vertical facial changes. During the 1940's and 1950's, surgeons concentrated on mandibular deformities and, thus, most procedures were performed on the mandible even when the basic deformity was found in the maxilla. This was partially due to the difficulties inherent in maxillary surgery such as vascularization and subsequent difficulty in mobilizing the maxilla. Until the advent of more sophisticated anesthetic techniques, antibiotics, and the biologic studies of Bell, complete maxillary surgery was uncommon and fraught with relapse. Hogeman was one of the first to correct the vertical maxillary deformities, but it was not until the work of Bell, Epker, and Schendel that the techniques were popularized.

Vertical Maxillary Excess: Long Face Syndrome

Vertical maxillary excess, or long face syndrome, was first recognized as such by Schendel and coworkers in 1976. Surgical correction of this deformity was based on a vertical impaction of the maxilla. Stability of this procedure, long thought to be prone to relapse, was demonstrated first in 1976 and was subsequently confirmed by numerous other studies. Skeletal open bite may or may not be a component of the long face syndrome and is one of its variants. This is due to a disproportion of the vertical excess, more vertical growth occurring in the posterior aspect of the maxilla versus the anterior, and an associated shortness of the ramus of the mandible.

Physical Findings

- Long vertical facial height, especially noted in the lower third
- Frequently narrow constricted alar bases
- Lip incompetence with an excessive interlabial gap
- Excessive gingival and upper incisor show at rest and during smiling
- Frequently an obtuse nasolabial angle >3110 degrees
- Retruded and vertically long chin
- Retrognathic mandible secondary to backward autorotation or to a true retrognathism

Cephalometric anaylsis

- Increased lower anterior facial height
- Smaller than normal SNA and SNB angles
- Larger than normal ANB angle (>3 degrees)
- Maxillary incisor show of >3 mm
- A steep mandibular plane angle
- Open antegonial angle
- Increased distance from the palatal plane to the occlusal surface
- Increased axial incisor angulation to the palatal plane >110 degrees
- A skeletal open bite may be present
- Retruded chin
- Usually decreased cranial base angle (basion-sella-nasion)
- Occlusion: Class II, occasionally Class I
- Frequent anterior open bite
- Constricted transverse maxillary arch, resulting in cross-bite
- Flat or accentuated Curve of Spee
- Frequent dental crowding

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Outline of Treatment

Presurgical Orthodontics

- Determine extraction versus non-extraction
- Orthodontic treatment
- Evaluate the amount of transverse maxillary deficiency; whether this can be corrected orthodontically or by multiple segmental mandibular osteotomies or by a separate transverse maxillary orthopedic expansion associated with surgical assistance
- Level and align the arches
- Eliminate dental compensation by aligning the teeth axially in their correct plane of alveolar bone
- No attempt to close any open bite should be done orthodontically

Surgical Orthodontic Assessment

- Assess facial aesthetics
- Definitive cephalometric prediction tracing
- Model surgery on a semiadjustible anatomic articulator with face bone transfer
- Splint construction

Surgical Plan

1. Maxillary Le Fort I osteotomy
   - ± multiple segments to correct any transverse or vertical problems
   - usual maxillary osteotomy is an impaction
2. Genioplasty
   - ± horizontal advancement
   - ± vertical reduction
3. Mandibular osteotomy
   - If auto-rotation alone is inadequate to correct the Class II malocclusion without significant posterior maxillary repositioning, the mandible should be advanced by bilateral sagittal split ramus osteotomies. Also, if any facial asymmetry is present, bimaxillary surgery will be required.

Vertical Maxillary Deficiency: Short Face Syndrome

Vertical maxillary deficiency was originally described by Hogeman but popularized after the article by Opdebeeck and Bell in 1978. Subsequent papers concerned the surgical correction of this condition by lengthening the maxilla vertically using interpositional graft material. Early reports were prone to high relapse with this procedure, but with rigid fixation, stability has been greatly improved.

Physical Findings

- Vertically decreased facial height
- Lack of maxillary incisor show with an edentulous look
- Frequently flat upper lip that appears short
- Concave facial profile with acute nasolabial angle <110 degrees
- Appearance of overclosure of the mandible with excessive projection of the chin
- Large gonial angles with appearance of large mass of muscles
- Wide alar bases

Cephalometric Analysis

- Decreased lower anterior facial height
- Decreased vertical height of the chin with frequent anterior projection
- A decreased distance from the occlusal plane to the palatal plane, indicative of shortening of maxillary alveolar height
- Increased maxillary incisor to palatal plane angle, indicative of proclined teeth
- Acute mandibular plane angle
- A skeletal Type III malocclusion
- Negative ANB angle
- A larger than normal to normal SNB angle
- Increased cranial base angle (basion-sella-nasion)

Occlusal Analysis

- Frequent deep bite
- Class II malocclusion to Class I
- Large transverse maxillary arch
- Frequently the mandibular teeth bite inside the maxillary dentition
- Frequent crowding of the mandibular dentition
- Reverse Curve of Spee

Outline of Treatment

Presurgical Orthodontics

- Level and align the arches
- Assess the need for extractions versus non-extraction treatment. More than likely with this malocclusion, extractions are not needed.
- The mandibular Curve of Spee may be accentuated. This needs to be leveled.
- The incisor should be placed over the alveolar bone with the correct axial inclination.

Surgical Orthodontic Treatment

- Reassess facial aesthetics
- Definitive surgical prediction tracing
- Model surgery on articulated dental casts by semiadjustable articulator
- Construction of occlusal splint

Surgical Plan

1. Maxillary Le Fort I
   - Frequently in one segment as most maxillas are sufficiently wide and level with this deformity
   - Vertical lenthening is most frequently done with an interpositional graft to improve the lip-to-tooth aesthetics
   - Occasional horizontal advancement of the maxilla to improve the profile and overjet relationship
2. Genioplasty
   - Vertical lengthening with interpositional graft to improve the lower facial height
   - Occasional retrusion of the chin if insufficient downward autorotation of the mandible has not occurred

Mandibular Surgery

Occasional mandibular advancement can be concomitantly done to improve overall facial projection, although this is infrequent with this type of deformity.

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CHAPTER 18

Maxillary Deficiencies

Jeffrey C. Posnick

A person with skeletal Class III malocclusion may exhibit either maxillary deficiency, mandibular excess, or a combination of the two. When maxillary deficiency is the primary morphologic problem, it may be in association with clefting (UCLP, BCLP, or ICP) or a more complex craniofacial syndrome (e.g., Apert, Crouzon, Pfeiffer, Kleeblatt–Schaeddel, or Binder's). Until the late 1960s, when Dr. Tessier developed the field of craniofacial surgery and Obwegeser introduced and Bell refined the Le Fort I osteotomy, the concept of surgical management of maxillary deficiency was of only theoretical interest. With these refinements in surgical technique maxillary deficiency is approached directly, rather than selecting a mandibular osteotomy as a camouflage technique to avoid "extensive" surgery on the upper jaw. Bimaxillary osteotomies are currently performed whenever indicated to give the best functional and aesthetic result.

Maxillary deficiency will result in a significant percentage of cleft lip and palate patients who undergo lip and palate repair in infancy and early childhood. The majority of patients born with a form of craniofacial dysostosis (e.g., Crouzon or Apert syndrome) will require a form of total midface advancement (monobloc bipartition, monobloc, or Le Fort III osteotomy) as part of their staged reconstruction. Those born with Binder syndrome will have maxillary and nasal deficiency requiring maxillary advancement and some form of nasal augmentation. Those born with Treacher–Collin syndrome or hemifacial microsomia will often have a degree of maxillary dysplasia requiring repositioning of the upper jaw in combination with osteotomies of the mandible and chin.

It has been estimated that in the Caucasian population, skeletal Class III malocclusion not related to clefting occurs in 1 to 2 percent of the population. Although the majority of patients with a Class III malocclusion will have mandibular excess, a component of maxillary deficiency/dysplasia is common enough that approximately 30 to 40 percent of those mandibular prognathisms will require maxillary osteotomies.

The purpose of this chapter is to review the physical findings of those patients presenting primarily with nonsyndromal maxillary deficiency and then discuss their treatment.

Physical Findings

- Poor lip to tooth relationship with edentulous look
- Short hypotonic retrusive upper lip
- Concave profile, acute nasolabial angle (>110 degrees)
- Overclosure of mandible with projection of chin button and accentuation of midface deficiency
- Narrow constricted alar base

Cephalometric Analysis

- Decreased lower anterior facial height with chin height usually decreased
- Decreased vertical maxillary alveolar height
- Mandibular incisor to mandibular plane angle indicating excessively proclined incisor teeth
- Maxillary incisor to sella-nasion plane angle indicating excessively proclined incisor teeth
- Skeletal Type III malocclusion: ANB angle (neg), SNA angle (acute), SNB angle (normal range)
- Increased freeway space (decreased vertical height)
- Mandibular plane angle usually acute

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Occlusal Analysis

- Anterior and labial crossbite
- Maxillary dental crowding
- Proclined upper incisors
- Normal or retroclined lower incisors

Outline of Treatment

Presurgical Orthodontics

- Assess need for extractions and need for surgical expansion of arch width prior to commencing treatment
- Align and level arches
- Eliminate maxillary crowding and dental compensation. Extract maxillary first bicuspids if space is needed, then retract maxillary canine and incisor teeth.
- Correct additional tipping, rotation, and spacing problems
- Eliminate mandibular crowding and dental compensation. Occasionally extraction of mandibular second bicuspid teeth is required to create space for correction of crowding, usually by advancing (proclining) lower anterior teeth. As part of the dental decompensation of occlusion, space is created to correct crowding. Levelling of the arch is usually accomplished by proclining the lower anterior teeth.

Surgical Orthodontic Reassessment

- Reassess facial aesthetics
- Definitive surgical cephalometric prediction tracing
- Model surgery on articulated dental casts
- Construct splints

Surgical Plan

Maxillary Le Fort I

- ± Midline split to increase transverse arch width
- ± Vertical lengthening with interpositional graft to improve lip-to-tooth aesthetics
- ± Horizontal advancement to improve profile and overjet relationship

Genioplasty

- ± Horizontal advancement to improve profile
- ± Vertical lengthening with interpositional graft to improve facial proportions

Maxillary Surgery

Historical Perspective

The Le Fort I maxillary osteotomy originated in Europe. In 1967, Cheever performed a unilateral Le Fort I osteotomy to remove a nasopharyngeal tumor. Later, Wassmund performed an osteotomy of the maxilla according to the fracture lines described by Le Fort in a patient with an anterior openbite deformity. Schuchardt was the first to separate the Le Fort I osteotomy at the pterygoid plates. Gillies and Rowe first described mobilization of collapsed maxillary segments in a patient with a cleft. Obwegeser showed that the down-fractured maxilla could be moved in any direction either as one unit or in segments. Bell, experimenting in dogs, demonstrated the blood supply, revascularization and bone healing that occurred with this procedure. A more complete history of the Le Fort I osteotomy can be found in an article by Freihofer.

Operative Technique

- Circovestibular mucousal incision from zygomatic buttress to zygomatic buttress is completed (leave thick flap of gingiva). Subperiosteal dissection of the maxilla is completed and retractors are placed for the osteotomy (Fig. 18.1A)
- With sterile pencil and calipers measurements are made and the osteotomy sites marked out (Fig. 18.1B)
- With a reciprocating saw, horizontal osteotomies are completed through the lateral, anterior, and medial maxillary walls (Fig. 18.1C)
- The septum of the nose is separated from the maxilla with an anterior nasal spine chisel (Fig. 18.1D)
- With the pterygomaxillary plate chisel, the pterygomaxillary sutures are fractured (Fig. 18.1F)
- With finger-pressure, the maxillary is down-fractured. It is further disimpacted with Tessier hooks (some surgeons prefer nasomaxillary disimpaction foreceps)
- A submucous resection of the nasal septum is completed if necessary to correct nasal obstruction (also to allow for maxillary intrusion) and, if indicated, the inferior turbinates are reduced (Fig. 18.1G and 18.1H)
- The disimpacted maxilla is brought into the prefabricated acrylic splint and the jaws are wired together (Fig. 18.1I)
- The desired vertical dimension is achieved, and a miniplate is placed across each zygomatic buttress, and each piriform aperture and secured with miniscrews (Fig. 18.1J)
- The jaws are unwired and the occlusion is checked
- If the maxilla has been either vertically lengthened or horizontally advanced a considerable distance, an interpositional graft (either autogenous bone or allograft) may be required (Fig. 18.1J)
- The circovestibular mucousal wound is closed

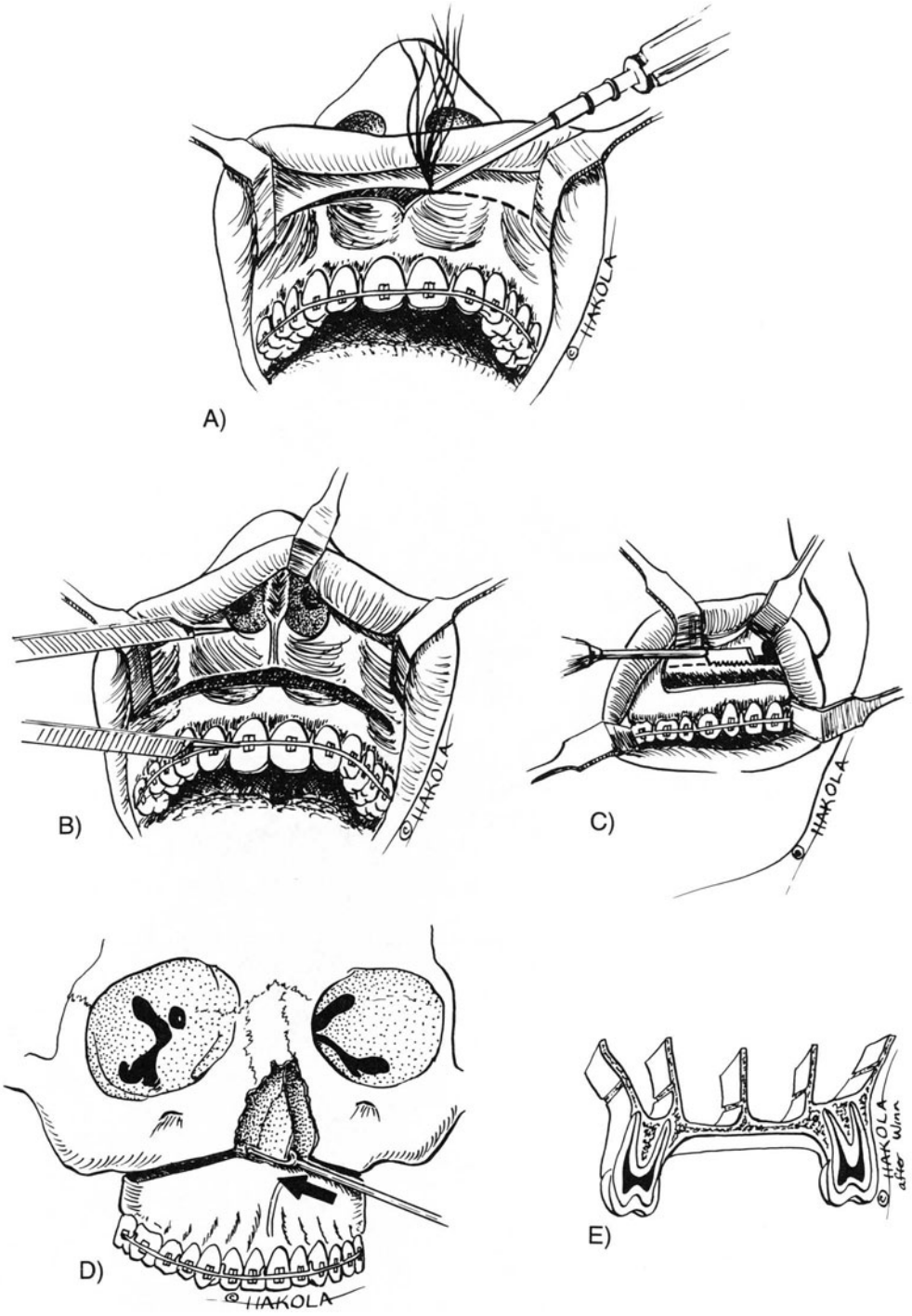

Figure 18.1. (A) A circovestibular mucousal incision from zygomatic buttress to zygomatic buttress is completed with either a knife or electrocautery. (A thick gingival cuff is maintained.) (B) After superiosteal dissection of the maxilla is completed, retractors are placed and the location of horizontal osteotomy is marked out with a caliper and pencil. (C) Horizontal osteotomies are completed with a reciprocating saw through the anterior, lateral, and medial maxillary walls. (D) The septum of the nose is separated from the maxilla with an anterior nasal spine chisel. (E) Schematic cross-section showing saw cuts.

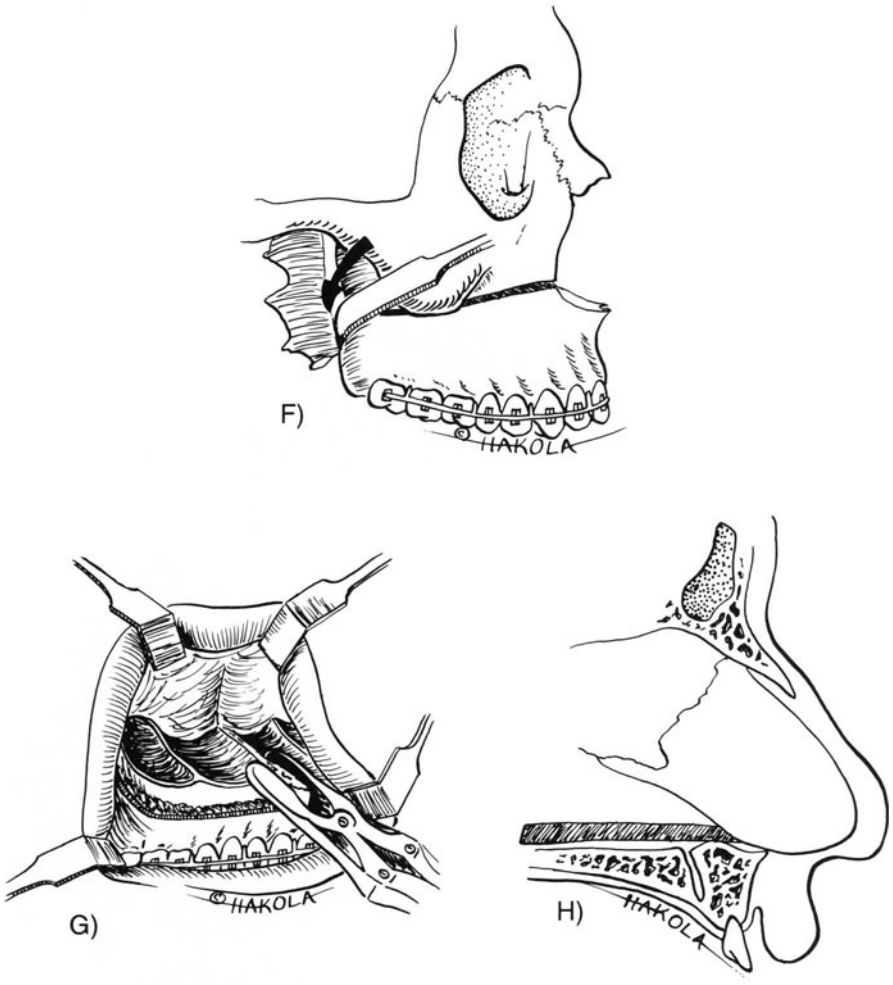

FIGURE 18.1. *Continued.* (F) The pterygomaxillary sutures are fractured using a pterygomaxillary plate chisel. (G) Once the maxilla is down-fractured and further disimpacted, a submucous resection of the nasal septum along the roof of the palate is completed (if indicated) to either relieve nasal obstruction or allow for necessary maxillary intrusion without causing buckling of the septum. (H) Diagram showing the amount of septal resection.

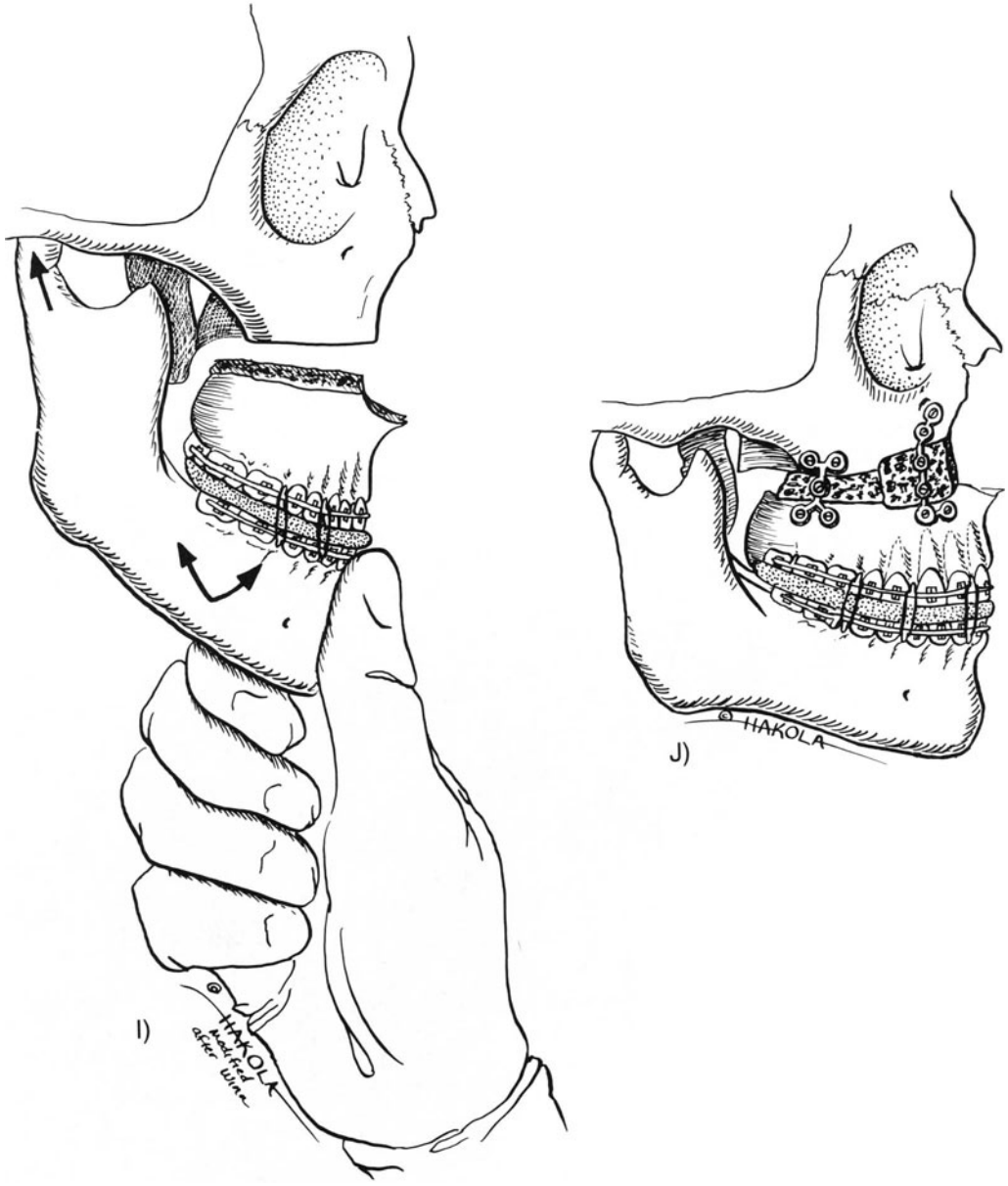

Figure 18.1. *Continued.* (I) Once the disimpacted maxilla is brought into the mandible through the prefabricated acrylic splint, the jaws are wired together in the preferred occlusion. Then the desired vertical dimension is achieved. (If the maxilla is being vertically lengthened, then an interpositional graft is placed.) (J) A miniplate is placed across each zygomatic buttress and each piriform aperture and secured with miniscrews. It is important to be certain that the condyles are properly seated in the glenoid fossa in the terminal hinge position.

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Chapter 19

Maxillary Deficiency: Unilateral Cleft Lip and Palate

Jeffrey C. Posnick

The correction of residual skeletal deformities of the unilateral cleft lip and palate (UCLP) adolescent challenges the ingenuity and skill of the maxillofacial surgeon. In theory, the UCLP patient will have undergone lip and plate repair in infancy and early childhood, followed by perialveolar fistula closure and bone grafting to the palate and alveolus while in the mixed dentition, then completing orthodontic treatment to close the cleft-dental gap in the region of the (often) congenitally missing lateral incisor tooth. However, this is often a goal not realized.

The central maxillofacial pathology in the UCLP adolescent patient presenting with Class III malocclusion is a degree of maxillary hypoplasia often combined with residual oronasal fistula, boney defects, and soft-tissue scarring. In addition, the maxillary lateral incisor tooth at the cleft site is usually congenitally absent, resulting in a cleft-dental gap.

Physical Findings

The adolescent with UCLP presenting for orthognathic surgery may have one or more of the following multiple residual clefting problems.

Maxillary Hypoplasia

The maxillary may be vertically short, resulting in an edentulous look. The occlusal plane is often canted. Arch width deficiency (transverse plane) resulting in lateral crossbites is a common finding. The maxillary dental midline may be shifted off the facial midline. The hypoplastic maxilla is retruded in the horizontal plane resulting in a concave midface profile, Class III malocclusion, and negative overjet. The greater and lesser maxillary segments generally vary in their degree of dysplasia resulting in discoordination of the maxillary arch.

Residual Oronasal Fistula

Residual labial and palatal fistula are often present despite previous attempts at closure. Buccal mucosa rather than keritonized gingiva may have been placed directly over the tooth-bearing, alveolar ridge as part of previous attempts at fistula closure.

Residual Boney Defect

Despite the preference for bone grafting the alveolus in the mixed dentition, the UCLP adolescent will often present with residual boney defects. Significant boney defects of the alveolus, hard-palate and floor of the nose along the cleft will result in discontinuity of the greater and lesser segments, inadequate boney support of cleft adjacent teeth, and an inferiorly displaced floor of the nose and nasal sill.

Cleft-Dental Gap

The lateral incisor is frequently congenitally absent at the cleft site. A rudimentary hypoplastic tooth may be present but with inadequate root development or with boney impaction in the cleft. The effect is often a dental gap at the cleft site between the central incisor and canine tooth.

Chin Dysplasia

The UCLP patient will frequently be a mouthbreather resulting from increased nasal air resistance. The increased nasal air resistance may result from a combination of factors including the presence of a pharyngeal flap, enlarged edematous turbinates, a cicatrated exter-

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nal nasal valve, and deviated nasal septum. The effect on skeletal morphology is often the development of a vertically long and retrognathic chin.

Mandibular Dysplasia

True mandibular prognathism in the Caucasian UCLP patient is rare. The need for mandibular osteotomies is limited to facial asymmetries, occlusal plane canting, and the occasional antero-posterior discrepancy.

Cephalometric Analysis

- Decreased anterior and posterior vertical maxillary alveolar height
- Increased or normal vertical chin height
- Skeletal Class III malocclusion: ANB angle (negative), SNA angle (acute), SNB angle (normal range)

Occlusal Analysis

- Maxillary dental midline noncoincident with mandibular dental midline
- Cleft-dental gap at cleft site
- Negative overjet and overbite relationship
- Lateral crossbite(s) from constricted arch width of maxillary lesser segment

Outline of Treatment

Presurgical Orthodontics

- Align and level mandibular arch, maxillary lesser and greater segments independently
- Eliminate maxillary crowding and dental compensation
- If indicated, extract maxillary supernumerary/rudimentary teeth at cleft site
- Correct additional tipping, rotation, and spacing problems
- Eliminate mandibular crowding and dental compensation
- Extraction of mandibular bicuspid teeth is only rarely indicated

Surgical Orthodontic Reassesment

- Reassess facial aesthetics
- Definitive surgical cephalometric prediction tracing
- Model surgery on articulated dental casts
- Construct acrylic splints

Surgical Plan

- Modified maxillary Le Fort I is generally required (see operative technique)
- ± Segmentalization with differential repositioning to assist closure of residual fistula, dead space, and cleft-dental gap, assist with coordination of arches and stabilization of segments
- Miniplate and screw fixation
- Autogenous bone grafting to palatal, nasal, alveolar, and interpositional defects
- ± Sagittal osteotomies of mandible to correct facial asymmetries, occlusal plane canting or anteroposterior discrepancies. Stabilization with bicortical screws
- Genioplasty
  - ± vertical reduction to improve lip seal
  - ± horizontal advancement to improve profile

Cleft Orthognathic Surgery

Historical Perspective

For effective management of the UCLP maxilla presenting with multiple residual deformities, both the classic Le Fort I osteotomy and previously reported techniques were modified by Posnick. His principal modification consisted of placement of soft-tissue incisions that allow direct exposure for dissection, osteotomies, disimpaction, fistula closure, bone grafting, and application of plate-and-screw fixation, but without risk of circulation injury to the dento-osseous-musculo-mucosal flaps. The increased visibility provided by this incision placement allows for the surgical closure of the cleft-dental gap through differential maxillary segmental repositioning. This method of approximating the maxillary segment for closure of the gap also closes the dead space where the cleft boney gap existed, and approximates the labial and palatal flap to allow for closure of recalcitrant oronasal fistula.

Operative Technique

- A maxillary vestibular incision is made from one zygomatic buttress to the other without the need of maintenance of labial pedicles (Fig. 19.1A)
- Parallel incisions are then made in the region of the residual labial oronasal fistula, separating the oral and nasal mucosa on each side of the cleft (Fig. 19.1B)
- The nasal and oral mucosa are also sharply incised on the palate to separate the mucosal layers and facilitate fistula closure (Fig. 19.1C)
- Undermining of the palatal mucosa and periosteum from the bone of the hard palate is carefully prevented because blood flows from these flaps to the bone and then the teeth of the lateral segments (Fig. 19.1C)
- Soft-tissue subperiosteal dissection exposes the anterior maxilla on each side. Horizontal osteotomies are carried out through the lateral, anterior, and medial maxillary walls with a reciprocating saw (Fig. 19.1D)
- The deviated nasal septum is separated from the maxilla with an anterior nasal spine chisel (Fig. 19.1E)
- The pterygomaxillary sutures are also separated with a pterygomaxillary chisel (Fig. 19.1F)

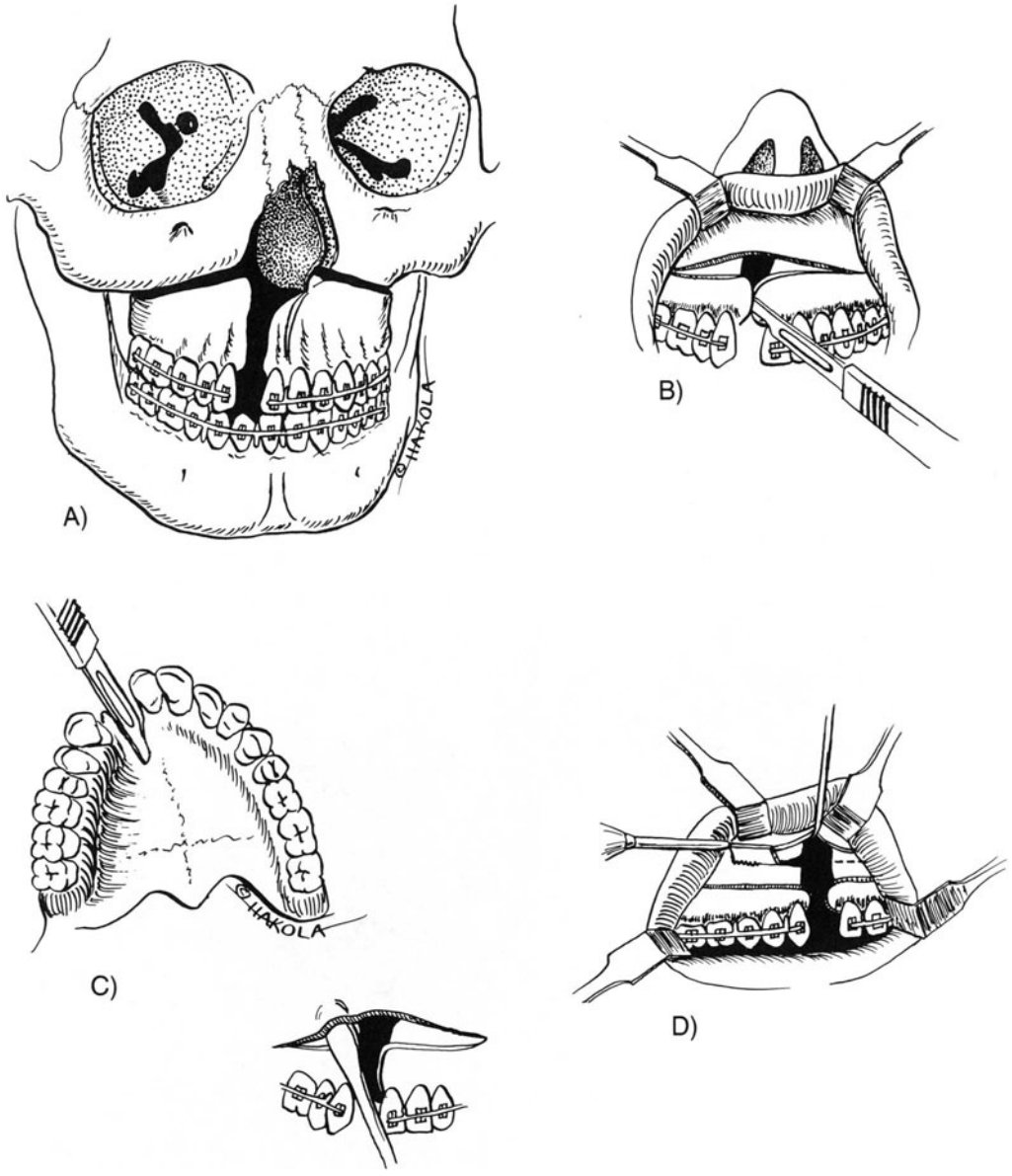

Figure 19.1. (A) A maxillary vestibular incision is made from one zygomatic buttress to the other. (B) An incision is then made in the region of the residual labial oronasal fistula, separating the oral and nasal mucosa on each side of the cleft. (C) The nasal and oral mucosa are also sharply incised on the palate to separate the mucosal layers and facilitate fistula closure. Soft tissue subperiosteal dissection exposes the anterior maxilla on each side. (D) Horizontal osteotomies are carried out through the anterior, lateral, and medial maxillary walls with a reciprocating saw.

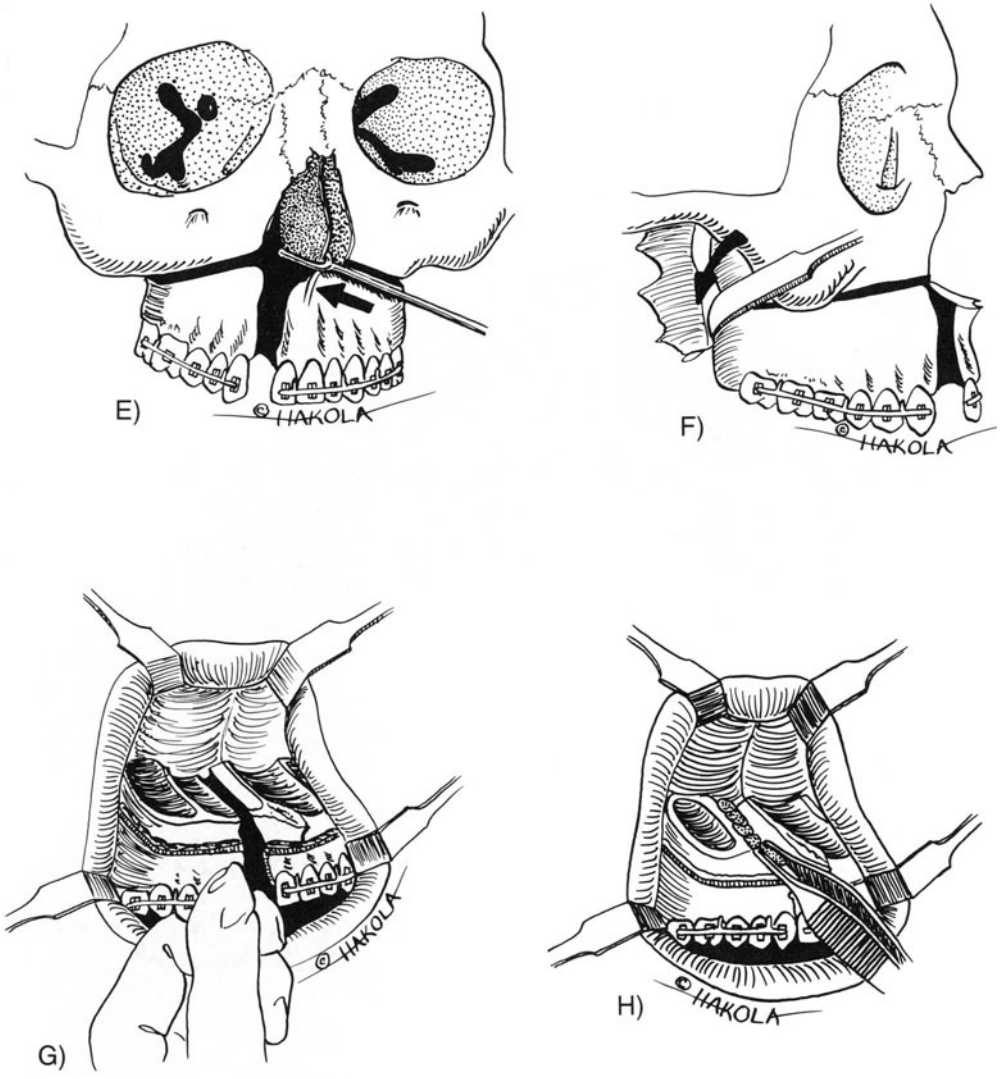

Figure 19.1. *Continued.* (E) The deviated nasal septum is separated from the maxilla with an anterior nasal spine chisel. (F) The pterygomaxillary sutures are also separated with a pterygomaxillary chisel. (G) The maxilla is down-fractured with finger pressure and disimpacted with Tessier hooks. (H) Cancellous bone graft is packed along the clefted palate and the floor of the nose beginning posteriorly and eventually filling the nasal sill.

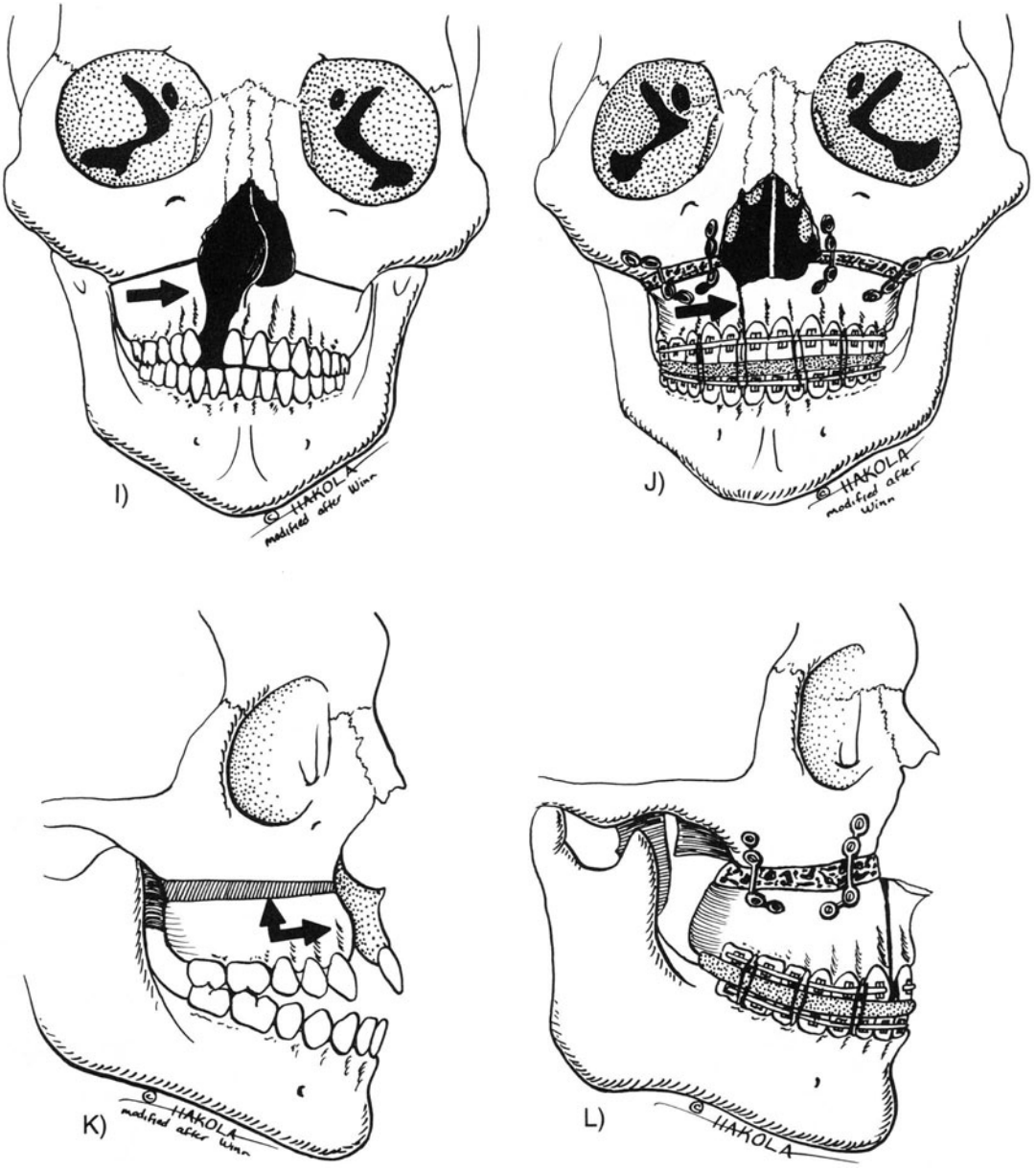

Figure 19.1. *Continued.* (I and J) The ideal vertical dimension is achieved at the maxillary osteotomy site and miniplates are applied at each zygomatic buttress and piriform aperture, as shown in a frontal view. (K) and (L) show a lateral viewpoint.

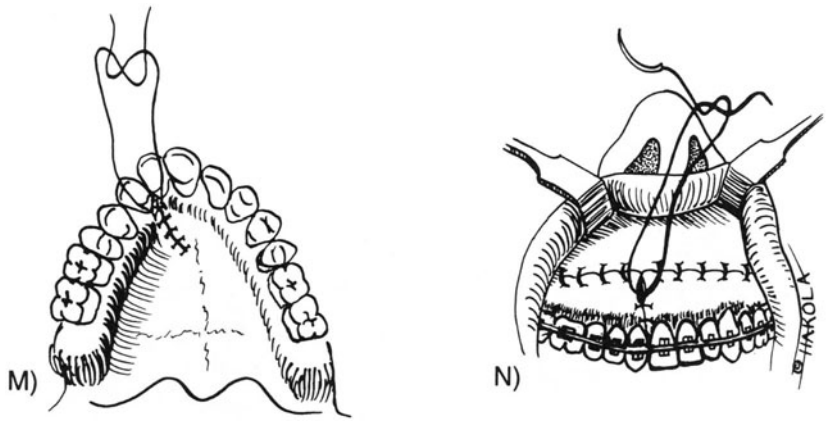

Figure 19.1. *Continued.* (M and N) With the cleft-dental gap surgically closed, the gingival mucosal flaps on the labial aspect and the mucosal flaps on the palatal aspect are approximated and sutured.

- The maxilla is down-fractured with finger-pressure and disimpacted with Tessier hooks. Rowe's disimpaction forceps are avoided as this would contuse the palatal mucosa (Fig. 19.1G)
- Vomer flaps are elevated and a submucosal resection of the inferior aspect of the Vomer and cartilagenous septum is completed to manage any septal deviation
- The enlarged, inferior turbinates are reduced, then the nasal mucosa is sutured for a water-tight nasal side closure of the fistula tract
- The down-fractured maxilla is generally in two segments as a result of the original bony cleft. If this is not the case and surgical closure of the cleft-dental gap is required, the segments are separated with a rotary drill. The maxillary segment are then aligned into the prefabricated acrylic occlusal splint
- The maxilla within the splint is then placed onto the mandible and intermaxillary fixation (IMF) applied
- The ideal vertical dimension, determined before the operation, is achieved at the maxillary osteotomy site and bone miniplates are applied at each zygomatic buttress and piriform aperture (Fig. 19.1I and 19.1J)
- The IMF is released and the occlusion checked
- Cancellous bone graft is packed along the clefted palate and the floor of the nose, beginning posteriorly and eventually filling the nasal sill (Fig. 19.1H)
- A strut of cortico-cancellous graft is interposed at the osteotomy site between the zygomatic buttress and piriform aperture on each side to fill the gap created by the maxillary advancement. Stabilization of the graft is with microplates and screws (Fig. 19.1K and 19.1L)
- With the cleft-dental gap surgically closed, the gingival mucosal flaps on the labial aspect and the mucosal flaps on the palatal aspect are approximated and direct suturing is carried out without the need for separation of the flaps from their underlying bone for advancement (Fig. 19.1M and 19.1N)
- If mandibular osteotomies are required, they are carried out as standard sagittal split osteotomies of the mandible
- If a genioplasty is required (e.g., vertical reduction and horizontal advancement), this is carried out in standard fashion.

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CHAPTER 20

Maxillary Deficiency: Bilateral Cleft Lip and Palate Deformity

Jeffrey C. Posnick

The bilateral cleft lip and palate (BCLP) adolescent that presents with residual cleft skeletal deformity may be the most challenging problem that the maxillofacial surgeon will face. The central pathology in this patient is maxillary dysplasia with hypoplastic lateral segments, a mobile premaxillary unit that may be either vertically elongated or shortened and horizontally retruded. Residual oronasal fistula, bony defects, and soft-tissue scarring are often additional problems. The maxillary lateral incisor teeth are congenitally absent in most patients, resulting in bilateral cleft-dental gaps.

Physical Findings

Adolescents with BCLP presenting for orthognathic surgery may have the following multiple residual clefting problems.

Maxillary Dysplasia

The premaxilla may be either vertically long, resulting in a gummy smile, or vertically short with an edentulous look. There may be a negative overjet, indicating a horizontal deficiency. In the transverse plane, the arch width of the lateral segments is generally deficient, with bilateral crossbites and a degree of horizontal deficiency with a Class III malocclusion.

Residual Oronasal Fistula

The BCLP adolescent will often have residual labial and palatal fistulas with loss of fluid through the nose while drinking and air leakage while speaking. Previous attempts at fistula closure may have ended in failure. Buccal mucosa rather than keritonized gingiva may rest adjacent to the cleft and surround the teeth next to the cleft.

Cleft-Dental Gaps

The lateral incisor teeth in the maxilla are frequently congenitally absent. Rudimentary teeth may be impacted within the cleft. The end result is often a dental gap at the cleft site between the central incisor and canine tooth on each side.

Residual Bony Defect

Despite previous attempts to bone graft the alveolar defect, the BCLP adolescent may present with residual bony defects through the alveolus, floor of the nose, and palate with a resulting mobile premaxilla secured only to the bony nasal septum.

Chin Dysplasia

The BCLP patient will frequently be a mouthbreather as a result of a chronic increase in nasal air flow resistance. The end result is a vertically long and often retrognathic chin.

Mandibular Dysplasia

True mandibular prognathism in the BCLP Caucasian patient is rare. The need for mandibular osteotomies should be limited to facial asymmetries, occlusal plane canting, and the occasional anteroposterior discrepancy.

Cephalometric Analysis

- The anterior vertical maxillary alveolar height may be either elongated or short
- Vertical chin height is generally increased
- A skeletal Class III malocclusion is frequent: ANB angle (negative), SNA angle (acute), SNB angle (normal)

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- The posterior vertical maxillary alveolar height is generally decreased

Occlusal Analysis

- Bilateral, lateral crossbites (frequent)
- Bilateral, cleft-dental gaps with absent lateral incisor teeth (frequent)
- A negative or neutral overjet and overbite (frequent)
- A constricted maxillary arch width (frequent)

Outline of Treatment

Presurgical Orthodontics

- Assess need for extractions prior to treatment: there must be adequate alveolar bone support for all retained teeth
- Align and level mandibular and maxillary arches. Each of the three maxillary segment must be aligned and levelled independently for their ultimate union and coordination with the mandibular arch at the time of orthognathic surgery
- Eliminate maxillary crowding and dental compensation
- Correct additional tipping, rotation, and spacing problems
- Eliminate mandibular crowding and dental compensation
- Extraction of mandibular bicuspid teeth is only rarely indicated

Surgical Orthodontic Reassessment

- Reassess facial aesthetics
- Definitive surgical cephalometric prediction tracing
- Model surgery on articulated dental casts
- Construction of acrylic splint(s)

Surgical Plan

Modified Maxillary Le Fort I
(See Operative Technique for Details)

- Maxillary Le Fort I segmental osteotomies
- Autogenous bone graft to interpositional defects and boney clefts
- Fixation with miniplates and screws
- Application of prefabricated acrylic splint

Mandibular Osteotomies

- ± Sagittal split osteotomies of the mandible
- Stabilization with bicortical screws

Genioplasty

- ± Vertical reduction for correction of lip incompetence
- ± Horizontal advancement to improve profile

Cleft Orthognathic Surgery

Historical Perspective

Steinkamm's first description, in 1938, of a maxillary Le Fort I osteotomy in a BCLP patient was that of a man who had previously undergone resection of the premaxilla. In 1974, Willmar reported on the complications associated with the Le Fort I osteotomy in both cleft and non-cleft patients. One of the 8 patients with BCLP in his group of 106 operated patients died following surgery. Henderson and Jackson, and others, have previously reported approaches to managing residual skeletal clefting problems. Posnick recently described a one-stage surgical approach for the management of the end stage residual clefting problems in the BCLP patient, his primary modification being incision placement that allows direct exposure for the necessary dissection, segment disimpaction and repositioning, a layered oronasal fistula closure, effective bone-graft placement, and application of miniplate-and-screw fixation but without flap compromise. His technique is dependent on a thorough understanding of the dento-osseous-musculo-mucosa flap blood supply. Preservation of the circulation to the lateral maxillary segments is through the palatal mucosa and periosteum. The circulation to the premaxillary segment is through the labial mucosa and periosteum. These flaps can be safely elevated without risking aseptic necrosis and the resultant loss of bone and teeth. This technique also permits simultaneous surgical closure of the cleft-dental gap on each side through differential maxillary segmental repositioning. The approximated maxillary segments also serve to close down the dead space at each cleft site and allow direct approximation of the labial and palatal mucosal flaps for successful closure of recalcitrant oronasal fistula (Fig. 20.1A and 20.1B).

Operative Technique

- The buccal incisions are in the depth of the vestibule and extend from the zygomatic buttress forward to the location of the residual labial oronasal fistula on each side, then down the mesial line angles of the canine teeth
- The premaxillary segment incisions are placed adjacent to the distal line angle of the incisor tooth on each side (Fig. 20.1C and 20.1D)
- The nasal and oral mucosa are sharply incised and separated on the palatal aspect of the premaxillary segment and of each lateral segment (Fig. 21C–E)
- Care is required to prevent any incision or disruption of the mucosa of the labial vestibule of the premaxilla, and to prevent undermining of the palatal mucosa or periosteum from the bone of the hard palate within

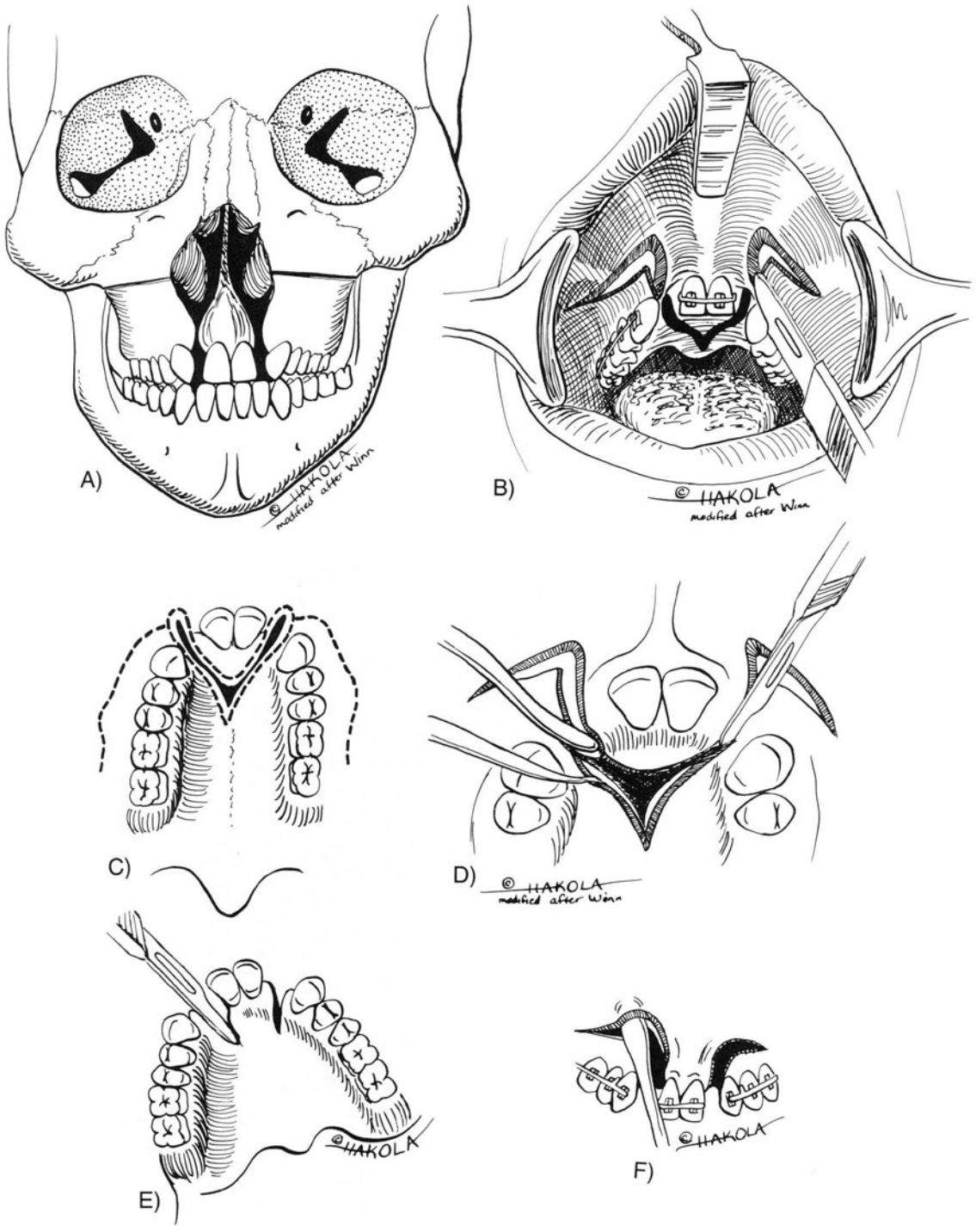

Figure 20.1. (A) Bilateral cleft palate abnormalities shown in a frontal view. (B) The labial-lateral vestibular mucosa incisions are made. (C) Planned incisions on the palatal aspect of the premaxillary segment. (D, E, and F) Soft tissue subperiosteal dissection provide direct exposure of the anterior maxilla on each side.

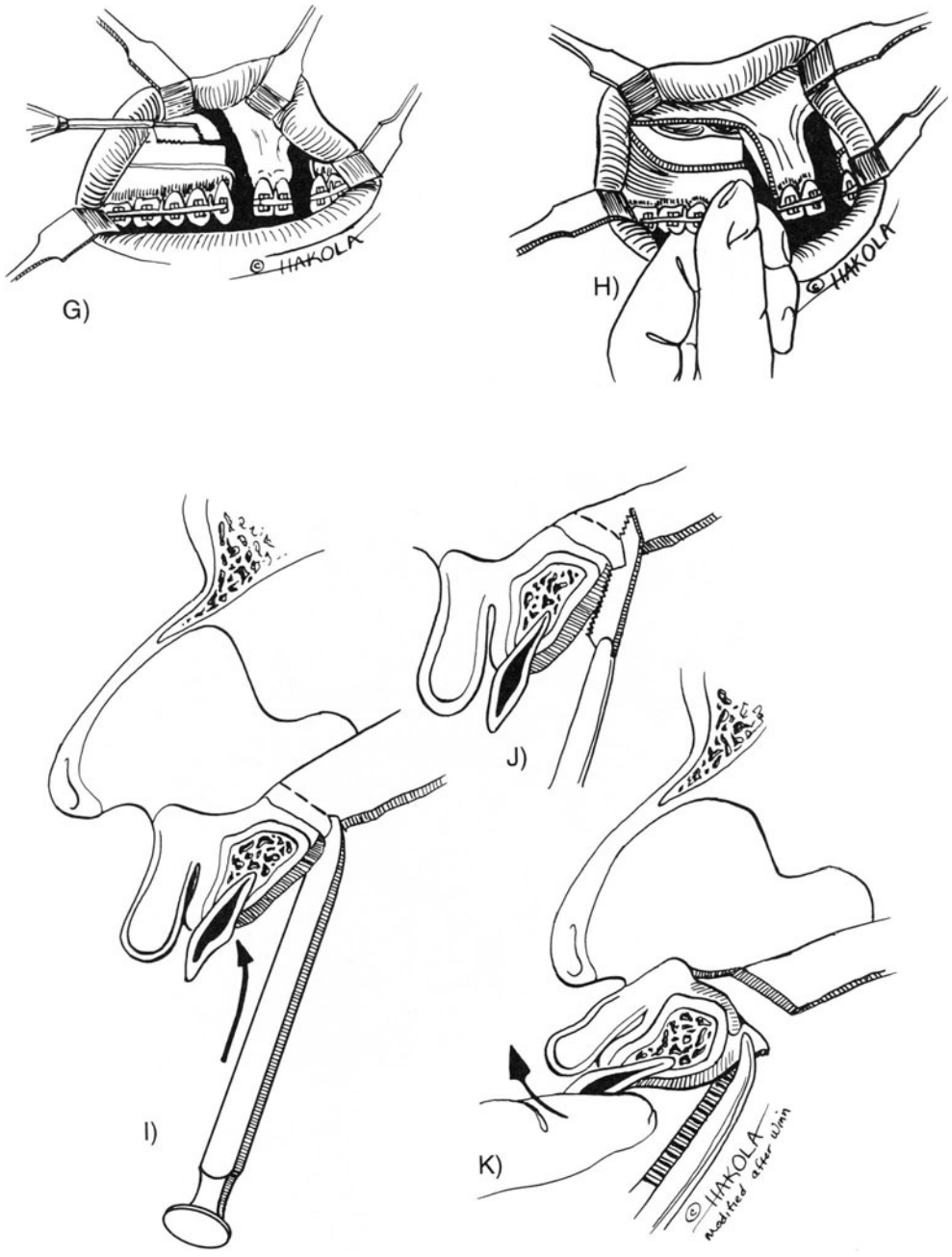

Figure 20.1. *Continued.* (G) Horizontal osteotomies are performed. (H) The maxillary lateral segments are down-fractured with finger pressure. (I, J, and K) The premaxillary segment is osteotomized from the palate with a reciprocating saw and out-fractured.

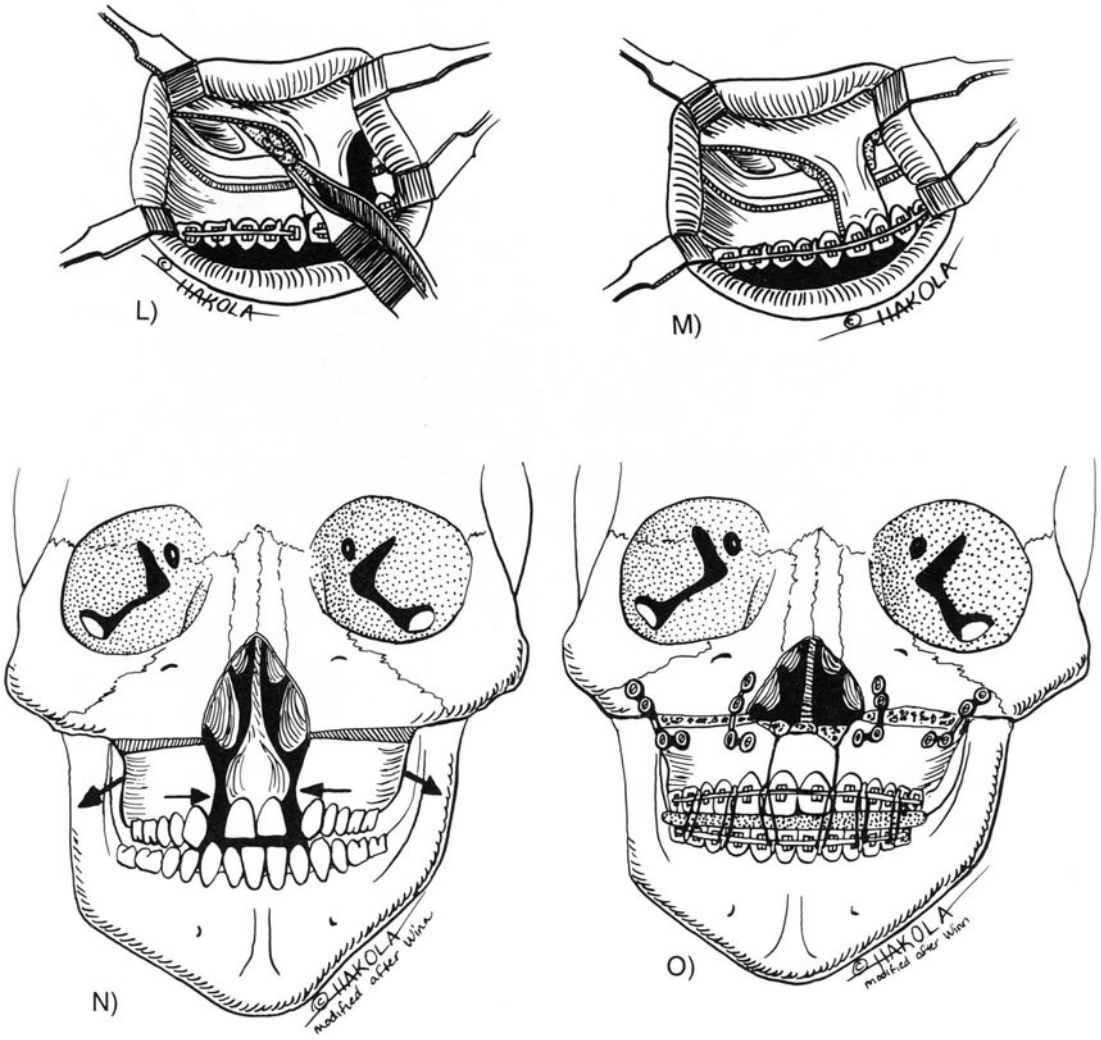

Figure 20.1. *Continued.* (L and M) Cancellous bone graft is packed along the clefted palate. (N and O) The ideal vertical dimension, determined prior to the operation, is achieved using cortico-cancellous bone struts along the bone gap between the zygomatic buttresses and the piriform aperture.

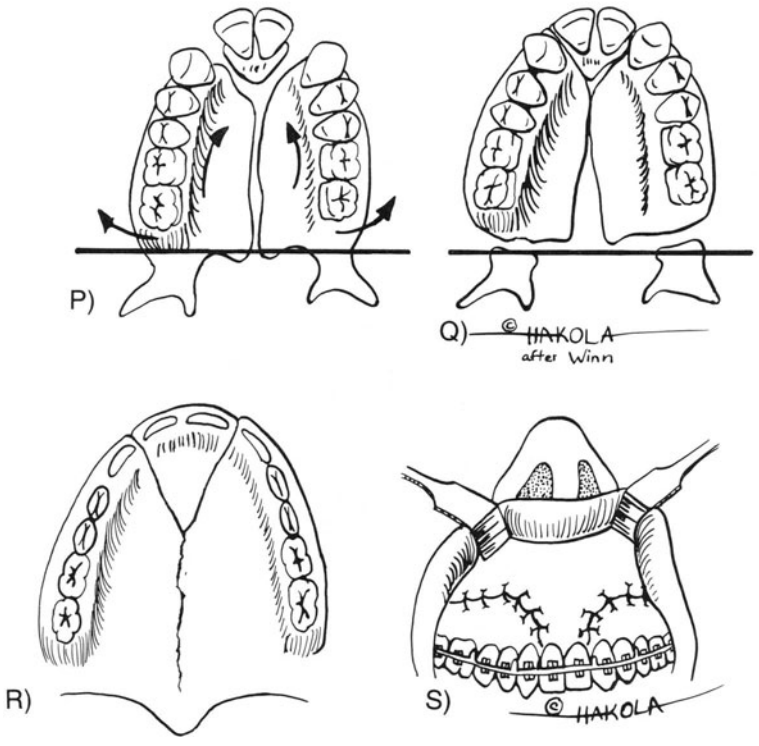

Figure 20.1. *Continued.* (P and Q) Degree of segmental repositioning of the palate is shown, which closes premaxillary/maxillary segment gaps. (R and S) The gingival and palatal mucosa flaps are approximated and directly sutured as needed without the need for rotation flaps.

either lateral segment. The blood that flows from these pedicles to the lateral and premaxillary segments is critical to each flap's survival

- Soft-tissue subperiosteal dissection provides direct exposure of the anterior maxilla on each side. Horizontal osteotomies are performed through the lateral, anterior, and medial maxillary walls with a reciprocating saw (Fig. 20.1F and 20.1G)
- Pterygomaxillary sutures are separated with a mallet and chisel
- The maxillary segments are down-fractured with finger-pressure, disimpacted with Tessier hooks, and freed of scar tissue for three-dimensional repositioning. If the vomer is fused to the lateral segments, then osteotomy is initiated before down-fracture (Fig. 20.1H)
- The premaxillary segment is osteotomized from the palate to avoid disruption of the labial vestibule mucosa from the premaxillary bone. This is done with a reciprocating saw or an osteotomed (Fig. 20.1I–K)
- A submucous resection of the inferior aspect of the Vomer and cartilagenous septum is completed and the inferior turbinates reduced as indicated. The nasal mucosa lining is sutured closed.
- The lateral maxillary segments are advanced and ligated into a prefabricated acrylic occlusal splint as is the premaxillary segment (Fig. 20.1M)
- The differential segmental repositioning approximates the labial and palatal mucosal flap without the need to separately advance local flaps for oral side fistula closure
- The maxilla through the splint is advanced on to the mandible and IMF applied.
- The ideal vertical dimension, determined before the operation, is achieved and bone miniplates are applied across the osteotomy sites at the zygomatic buttress and piriform aperture on each side (Fig 20.1 N–R)
- The maxilla is down-fractured with finger pressure and disimpacted with Tessier hooks. Rowe's disimpaction forcego are avoided as this world confuse the palatal mucosa (Fig. 20.1G)
- The IMF is released and the occlusion is checked
- Cancellous bone graft is packed along the floor of the nose and clefted palate on each side (Fig. 20.1L)
- Cortico-cancellous bone struts are interposed along the bone gap at the (advanced) anterior maxilla between the zygomatic buttress and piriform aperture

on each side. The grafts are secured with microplates and screws (Fig. 20.1O)
- With the cleft-dental gaps surgically closed, through differential segmental repositioning, the gingival and palatal mucosa flaps are approximated and directly sutured without the need for rotation flaps (Fig. 20.1R and 20.1S)
- If mandibular, bilateral split osteotomies and/or genioplasty are required, these are completed in standard fashion

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CHAPTER 21

Traumatic Deformities and Reconstruction of the Temporomandibular Joint

Jonathan S. Jacobs and David Mendes

Temporomandibular joint (TMJ) disorders and their treatment have been the source of ongoing controversy for decades. Debate concerning pathologic processes and treatment modalities has led to a wide array of treatment options. However, results have been difficult to evaluate, and a clear consensus regarding the approach to disorders of this unique structure has yet to be reached.

Anatomy/Embryology/Physiology

The TM joint is the articulation between the mandibular condyle and the squamous portion of the temporal bone. It is a synovial, diarthrodial, fully movable joint.

It is first noted in the eighth week of gestation as condylar and temporal blastemas or mesenchymal condensations. These are ossified between the 9th and 10th weeks of gestation, and condylar cartilage appears.[1] Meckel's cartilage, the embryological core of the mandible, is wrapped in membranous bone. It gives rise to the incus and malleus as well as the mandible. Meckel's cartilage has no role in the development of the temporomandibular joint.[2]

The mandible's ossification center (the first to be formed in the body)[3] is evident at 6 weeks of gestation. The articular disc forms from a condensation of mesenchymal cells between the condylar and temporal blastemas. The anterior portion is derived from the condylar blastema and the posterior portion from the temporal blastema. The medial portion of the disc derives from the tendon of the lateral pterygoid muscle. The disc achieves its mature, genetically determined shape by 14 weeks of gestation. The central region (intermediate zone) is thin and the periphery is thicker. At the 12th week of gestation, the lower joint compartment forms by cavitation and cell death and enzymatic breakdown. The upper joint space is formed at 14 weeks. The process requires muscular activity, and in its absence, an abnormal, fused joint is formed.

The mandibular condyle is not covered by hyaline cartilage, as are the articular surfaces of other joints. Rather, its surface is covered by avascular fibrous connective tissue that contains cartilage cells. The condyle consists of cancellous bone covered by a thin layer of cortical bone.

The maxillary and mandibular dentition influence joint movement and position, as they determine the end point of closure. Thus, many treatment modalities have been devised that use occlusal repositioning devices directed at joint stabilization.

The meniscus, or articular disc, is interposed between the condyle and the temporal bone, separating the joint into upper and lower joint spaces (Fig. 21.1A and 21.1B). The precise role of the meniscus remains the subject of discussion. Possible functions include that of a hydraulic shock absorber, protecting the condyle and its articular cartilage during translation, joint lubrication, and a possible role in the self-repair mechanisms.[4] The articulation of the mandibular condyle with the articular disc forms the ginglymoid lower joint: the simple hinge disc-condyle complex. This disc-condyle complex articulates with the temporal bone to form the upper, arthrodial, portion of the TMJ. Sliding movement in the upper joint space is translation. In the adult, the average interincisal opening is approximately 40–50 mm. The first 20–25 mm of opening are allowed by hinge activity, and the remaining 15–20 mm by translation antero-inferiorly along the articular eminence.

The condyles are elliptically shaped, measuring approximately 15–20 mm in the medio-lateral axis and 8–10 mm in the antero-posterior axis. The long axis of

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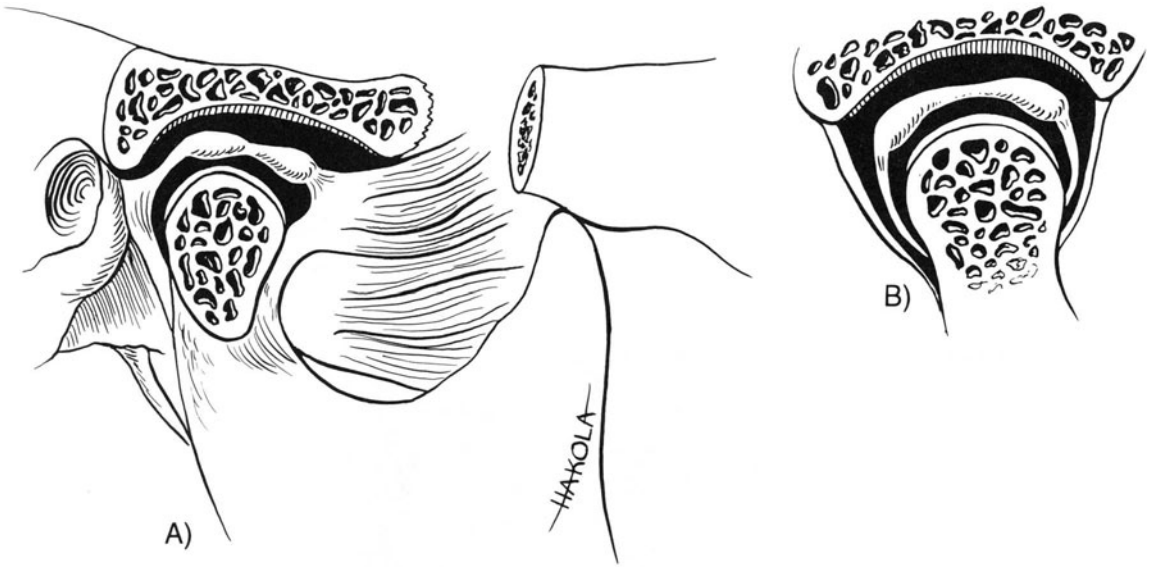

FIGURE 21.1. (A) The temporomandibular joint is separated into upper and lower joint spaces by the articular disc (meniscus). Lower joint space movement is rotational. Upper joint space movement is translational. (B) Note medial and lateral attachments of the disc distinct from the joint capsule.

the condyle is oriented perpendicular to the plane of the mandibular ramus.

The articular surface of the temporal bone lies anterior to the tympanic bone and is composed of a concave glenoid fossa and the convex articular eminence anteriorly. During translation the path traveled by the condyle is guided by the articular eminence.

The joint capsule, composed of fibrous connective tissue, attaches superiorly to the inferior aspect of the zygomatic arch and inferiorly to the condylar neck and periosteum (Fig. 21.1B). The capsule fuses with the meniscus, except medially and laterally, where it meets the condylar neck.

The temporomandibular (TM) ligament is that thickened portion of the capsule extending antero-laterally. The TM ligament reinforces the lateral capsule.

The articular disc includes a thin central region and thickened regions anteriorly and posteriorly. The anterior band conforms to the articular eminence and attaches to the lateral pterygoid muscle. Inferiorly on the medial and lateral sides, the meniscus attaches to the condyle.

Posteriorly, the bilaminar retrodiscal pad is formed by a superior fibroelastic lamina that attaches to the tympanic plate of the temporal bone, and an inferior lamina consisting of non-elastic connective tissue that attaches to the condyle. Between these two lamina exists a highly vascularized, highly innervated zone of loose areolar tissue. As the condyle achieves maximal translation anteriorly, the superior lamina is stretched, stabilizing the disc on the superior surface of the condyle and balancing the action of the inferior head of the lateral pterygoid muscle, whose pull is in an anterior medial direction. As the mandibular condyle is drawn posteriorly from its anteriorly translated position, the superior belly of the lateral pterygoid muscle, dormant during opening, pulls against the condyle and the retrodiscal pad. Thus, the condyle is stabilized against the articular eminence during closure and the meniscus is pulled into its anteriorly rotated position on the condyle[48] (Fig. 21.2).

The articular disc and its supporting attachments divide the joint into upper and lower joint spaces. The shape of the articular disc conforms superiorly to the glenoid fossa and inferiorly to the mandibular condylar head.

Mechanics

The muscles of mastication and the surahyoid muscles combine to effect three types of movement: rotation (hinge), translation (gliding), and a combination of rotation and translation. Rotation and slight translation occur in the lower joint space. Translation is effected in the upper joint space. In the retruded position, the retrodiscal pad covers the superior aspect of the condyle. The initial phase of opening is characterized by rotational movement. During rotation, the posterior band moves posterior to the condyle, and the condyle engages the intermediate zone of the disc and articulates with the articular eminence. The disc-condyle complex then shifts with further opening: the disc follows a path along the articular eminence, lagging behind the condyle. In the third terminal phase of maximal opening, the condyle may rotate under the anterior band.

As the condyle is displaced anteriorly, the retrodiscal pad fills with blood, thus filling the space evacuated by the moving condyle. As the condyle retrodisplaces during closure, the condyle comes in contact with the fossa and blood is evacuated from the retrodiscal pad. The superior head of the lateral pterygoid comes into play

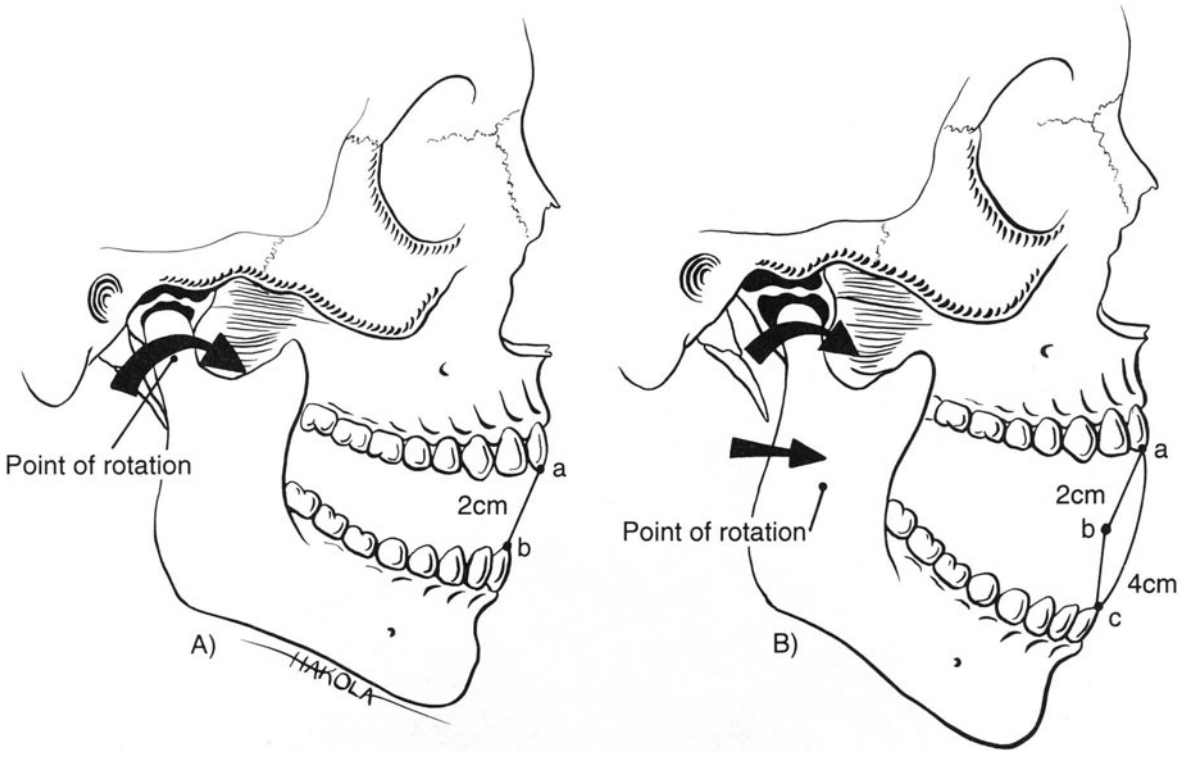

FIGURE 21.2. (A) The articular disc is stretched forward with the action of the external pterygoid muscle. The retrodiscal tissues allow this stretch and return the disc to the fossa in closing. (B) Note the allowable range of motion in pure hinge a–b and with combined hinge, translation a–b.

during closure to stabilize the disc. The mechanism of disc movement is not clearly understood. The muscle that inserts on the disc's anterior band is the superior head of the lateral pterygoid, which is not active during opening[6] (Fig. 21.2B).

Diagnosis

Successful intervention for TMJ disorders requires a high degree of accuracy in establishing diagnoses. Analysis of a patient's signs and symptoms is directed toward achievement of an anatomic diagnosis. Intervention can be more intelligently planned if a specific pathologic process, with its possible etiologies, can be localized to a specific anatomic site. Disorders associated with interference with normal functioning of the articular disc lead to degenerative changes in the joint. Degenerative changes occur when functional demands are not satisfied by the adaptive response of the articular tissue. Common etiologic factors in degenerative TM disorders include trauma and aberrant function that overloads the joint, as well as internal derangement or disc malposition.[6]

The differential diagnosis of TMJ disorders is difficult. The surgeon is obliged to discern between processes that directly involve the joint and those that involve muscular structures. It is in the realm of joint disorders that the surgeon may be able to contribute. Muscular problems are less likely to be amenable to surgical therapy.

History

Patients' most frequent complaints include pain, joint noise, or clicking, and limitation of range of motion of the joint. Pain is most often the presenting symptom. The point of onset of the pain is important. More acute problems can generally be managed with simpler therapies. Was the pain related to trauma? Can the patient identify the location of pain? Patients suffering from joint derangements indicate the joint as the focal point of the pain, whereas patients suffering from muscular disorders often point to various areas associated with muscular structures as the source of their discomfort. The character of the pain, whether constant or intermittent, is revealing. Joint pain is always present, but muscular pain is not. Joint pain is often worse in the afternoons, muscular pain worse upon awakening. This may reflect a habit of nocturnal bruxism. The time of day during which symptom exacerbation develops may correlate with periods of increased physical or emotional stress. Joint pain is aggravated by mandibular activity, such as talking or mastication.

Joint noise, in the form of clicking or crepitation, requires further analysis. Clicking is the result of subluxation and relocation of the disc's posterior band (Fig.

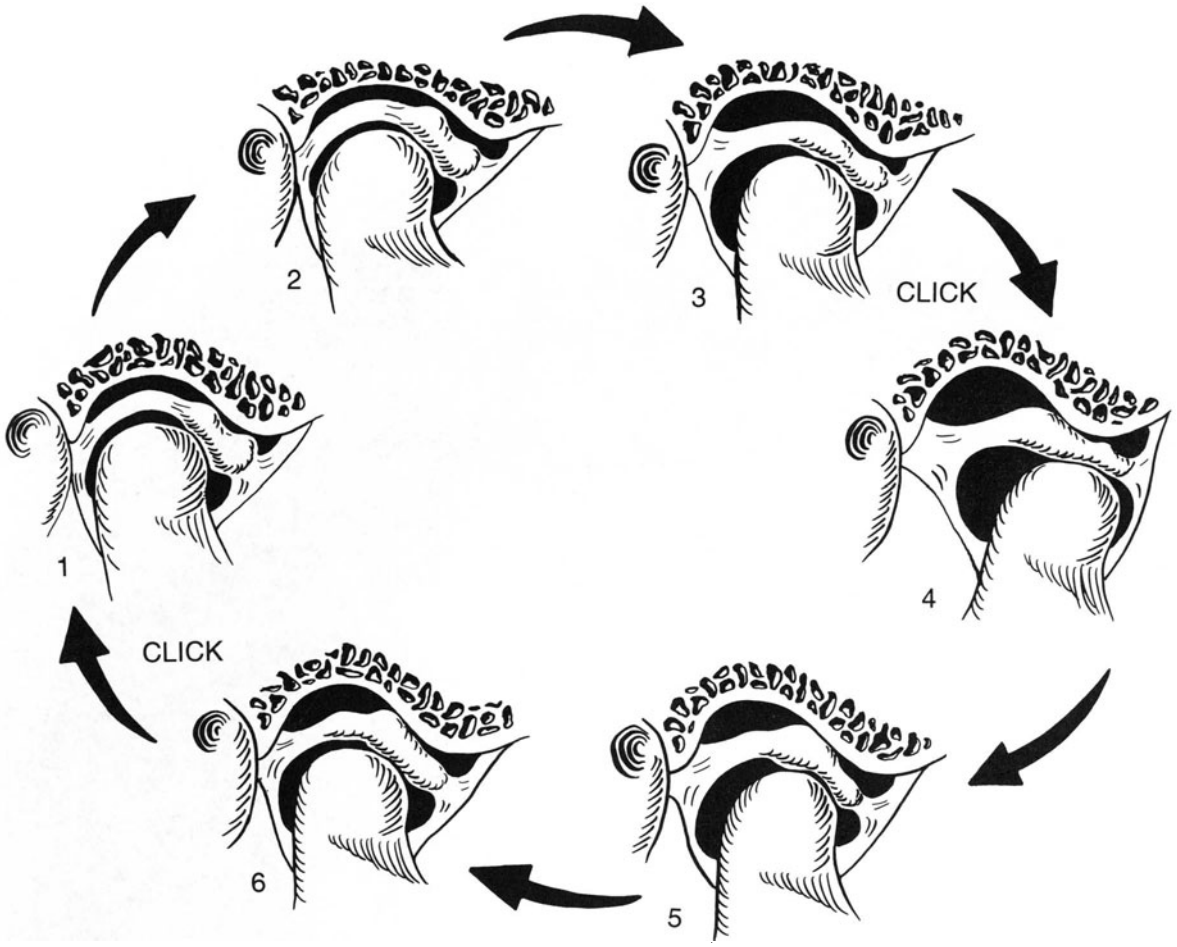

Figure 21.3. A click occurs when a subluxed posterior band of the articular disc returns to its anatomic position during translation 3–4. A reciprocal click may be felt as this band resubluxes in closing 6–1.

21.3). If clicking occurs only on awakening, this may be related to nocturnal bruxism. Pain associated with clicking is often noted in patients with internal derangement of the joint. It is not uncommon to have a patient report a crescendo of pain reaching a climax at the point when the click is felt, and for the discomfort to subside subsequent to the click. Crepitus relates to grinding of surfaces, as when the shock absorber effect of the articular disc is lost after perforation or disruption (Fig. 21.4). Calcification of the articular disc may also result in crepitus. Frequently, there is a history of clicking, a silent period with limited motion and then the advent of crepitant noise, indicating that perforation has probably occurred. With disc derangements, joint mechanics are affected. Should irreducible anterior malposition of the disc occur, "closed lock" with limitation of opening may occur. This is a more acute phenomenon then limitation of motion related to muscle function.

Toothaches, headaches, earaches, and neck aches are nonspecific symptoms described by patients with TM

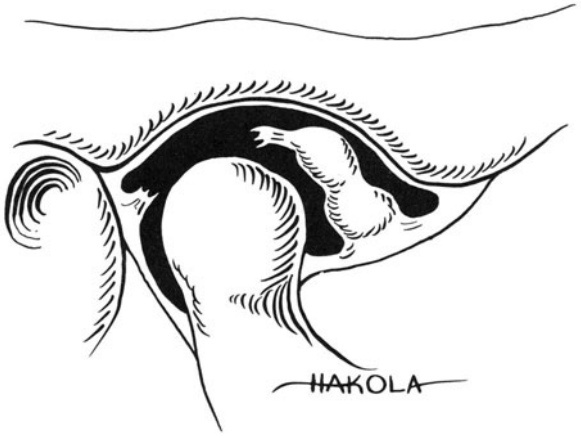

Figure 21.4. Crepitance and eventual degenerative change in the condyle may occur after perforation of the posterior attachment. When this condition is longstanding, the fibrocartilagenous covering of the condyle may be lost.

disorders. Pain may radiate to the neck and shoulders. These symptoms require appropriate investigation before they can be attributed to TMJ pathology.

Physical Examination

Mandibular range of motion should be measured. Normal opening is in the range of 40–45 mm, with normal lateral and protrusive ranges of approximately 10 mm. Normal opening is characterized by smooth, symmetrical movement of the mandible without deviation as it travels through its initial hinge, and subsequent translatory phases. The mandible may deviate to the affected side if there is disc subluxation and clicking. If there is a closed lock phenomenon with disc dislocation, range of motion will be limited and there will be deviation to the affected side. There will also be deviation with protrusion of the mandible, decreased range of motion on lateral movement (on the affected side), and possibly, pain. Patients with muscular disorders will demonstrate limited range of motion with deviation on opening but not necessarily on lateral movement. Protrusion is midline. Tenderness on palpation may be present and localized to the joint both laterally and through the external auditory canal.

Palpation and auscultation are useful in detecting joint noise. Clicking may represent disc subluxation caused by muscle spasm, which is reversible, or may represent disc subluxation, with injury to the retrodiscal pad. Forty percent of the population is thought to experience clicking,[5] and the significance of this is unknown.

Solitary opening clicks indicate that the disc is forced anteriorly, but reduces. Reciprocal or closing clicks can occur during retrusive motion, after an opening click (Fig. 21.3). If the disc is malpositioned anteriorly, the condyle travels anteriorly, its superior surface in contact with the posterior band rather than with the articular disc. As the condyle translates at the articular eminence, it reduces into the intermediate zone of the articular disc. With closing, the disc-condyle unit moves physiologically until the condyle retrodisplaces to a position under the posterior band, with the disc resuming its abnormal anteriorly dislocated position. Timing of the click in the joint motion cycle depends on the tightness of the meniscal attachments. As these stretch, the opening click occurs later in the cycle. With progression of the problem, a situation develops whereby the meniscus becomes caught anteriorly, and is irreducible. This is the "closed lock" phenomenon. The anteriorly dislocated, irreducible meniscus constitutes a mechanical obstruction and a cause of limited range of motion.

Crepitus signifies disease that has progressed to the point where irregular surfaces are grinding against each other. It occurs with disc perforation, and may also be found with disruption of meniscal attachments or adhesions within the joint. Crepitus may also be seen in cases of disc calcification.

A complete dental and occlusal examination is important. Bruxism is suggested by wear facets and tooth mobility or tenderness. The occlusion should be evaluated, including angle classification. The significance of malocclusion in TMJ disorders is controversial, but certain conditions are thought to have some bearing on the condition. These include incisal contacts that drive the mandibular condyle posteriorly, as in Class II Division 2 malocclusion, or missing posterior teeth. Prognathic, apertognathic, or long face syndrome patients are thought to be at risk for developing TMJ disorders. The masticatory musculature should be carefully inspected and palpated for evidence of asymmetry, hypertrophy, spasm, or tenderness. This should be combined with an examination of head posture, the neck and its range of motion, as well as other evidence of muscle spasm.

Imaging

Joint injuries may be the most common cause of meniscal internal derangement. These precede more severe degenerative conditions. Therefore, early diagnosis and intervention may avert late disability and suffering.[7]

Conventional Radiography

Screening of the craniomandibular complex is possible with antero-posterior, open mouth, jaw protruded and submental-vertex views. A panoramic mandibular film is most useful. The mandibular coronoid processes, condylar size, morphology, and symmetry can be examined. Decreased condylar size may be associated with advanced internal derangement. Cephalometrically corrected closed and open mouth lateral tomograms provide for detailed examination of condylar and glenoid fossa osseous anatomy, as well as analysis of condylar position within the glenoid fossa. No inference as to joint space or soft tissue disease should be made from these films.

Arthrography and Videofluoroscopy

Single (lower joint space) and two-compartment (upper and lower joint space) arthrographic techniques are excellent in detailing meniscal pathologic anatomy and displacement.[5,8] The concomitant application of videofluoroscopic study permits accurate assessment of joint dynamics as well as arthrographic interventional maneuvers. Mechanical distention of the temporomandibular joint space may effect lysis of intracapsular adhesions and liberate a tethered meniscus from the condyle or glenoid fossa. The utility of interventional arthrography has been demonstrated in the shoulder and wrist joints. The technique is limited by the patho-

logic condition of the joint space within which the procedure is performed. The capsule must be intact.

Computed Tomography

Computed tomography has become less useful in recent years with the increased reliance on improved arthrographic technique and the advent of magnetic resonance imaging. Computed tomography provides a good picture of the boney anatomy, and three-dimensional computed tomography may be useful in outlining craniofacial dysmorphology and facilitating surgical treatment planning. However, computed tomography cannot contribute to morphologic analysis of the intracapsular soft tissue abnormalities that are so significant in this region.

Magnetic Resonance Imaging

Magnetic resonance imaging is noninvasive, does not possess the disadvantage of ionizing radiation, allows examination in a multitude of planes, and is highly accurate in its representation of soft tissue. Fast scanning techniques are available for dynamic evaluation of the temporomandibular joint. Arthrography, however, is superior to magnetic resonance in detecting adhesions[9] and meniscal perforations. Because it is painless and noninvasive, magnetic resonance imaging has become the favored tool for analysis and determination of disc position.

Arthroscopy

Athroscopy as a diagnostic and therapeutic modality has gained popularity in recent years. Arthroscopy is less invasive than arthrotomy, and therefore, has been explored as a preferred alternative technique in selected TM disorders. Indications for TMJ arthroscopy include the need to examine the joint in cases in which less invasive diagnostic efforts fail to help the surgeon arrive at appropriate decisions regarding treatment.[10–12] Operative TMJ arthroscopy, thus far, has found more limited application in the treatment of posttraumatic disorders, although there are reports of symptomatic relief in cases of internal derangement following arthroscopic arthrolysis, lavage, and disc manipulation.[8] Interestingly, it appears that patients with closed lock disc derangement seem to benefit most, at least over the short term.

Myofacial Pain Dysfunction Syndrome

Myofacial pain dysfunction (MPD) syndrome is a catch-all designation that comprises a constellation of symptoms including:

1. Preauricular pain,
2. Masticatory muscle tenderness,
3. Lack of joint tenderness,
4. Limited mandibular opening,
5. Occasional clicking, and
6. Normal radiographic findings.

The causes of these symptoms are multiple. Malocclusion (in the form of mandibular overclosure or occlusal prematurity) associated with long-term microtrauma to the joint (as with bruxism) have been thought to be important in the etiology of the syndrome. Psychological factors, such as anxiety, have also been associated with MPD symptomatology. As a consequence, spasm of the lateral pterygoid muscle or deep posterior masseter may develop. Therapeutic efforts have been directed at increasing the posterior vertical dimension of occlusion by orthodontic or prosthetic means to alleviate the consequences of overclosure. Restricting mandibular opening to the initial hinge phase minimizes lateral pterygoid function. Splints have been designed to alter and condylar relationship to the glenoid fossa by disarticulating the dental occlusion and displacing the condyle caudally. The muscle spasm may be alleviated, and it is theorized that an anteriorly displaced meniscus may return to its physiologic position atop the mandibular condyle. It is important to determine the position of the articular disc prior to instituting therapy. In the face of internal derangement of the joint, occlusal adjustments, with the matching of centric occlusion to centric relation, drives the condyle posteriorly behind the meniscus, thereby exacerbating the condition. Secondary modalities used in the treatment of MPD include counseling, physiotherapy with midline hinge axis exercises, biofeedback, behavior modification, soft diet, heat, diathermy and massage, ultrasonography, and antiinflammatory agents and muscle relaxants.[3,6,8,13,14]

Internal Derangement

Internal derangement of the TM joint usually implies an anterior malposition of the meniscus, often with condylar malpositioning in the postero-superior direction. The retromeniscal pad is damaged, and may be disrupted. The patient may complain of preauricular pain, clicking and other less specific symptoms such as headache, earache, or neck ache. There may be a history of trauma, or of orthodontic treatment. Physical examination should be directed at the detection and characterization of the range of motion of the mandible, both in the vertical and lateral directions, of joint noises and clicks, including their timing within the joint cycle, and precise localization of pain and tenderness. Dental and occlusal examination are performed. Prognathism, long face syndrome and open bite conditions may be associated with TM dysfunction. Imaging with arthrographic or magnetic resonance techniques confirm the diagnosis.

Treatment

Conservative

If clicking is present early in the opening phase of the cycle, splinting is performed to disarticulate the occlusion and attempt to recapture the subluxing meniscus by positioning the condylar head beneath the meniscus. Periods of up to 3 months of splinting may allow the retrodiscal pad to recover from the stretching present during the period of internal derangement. Some patients, in whom the condylar head rests against the highly innervated retrodiscal pad, will suffer lateral pterygoid spasm, necessitating the use of muscle relaxants and other secondary maneuvers. There is some question as to the ability of the disc to return to a stable anatomic position. Perhaps the majority of patients become comfortable while functioning with a subluxed disc. This may be due to condylar remodeling.

Surgical

Surgical treatment is indicated in the face of failure of conservative therapy to relieve joint pain and clicking and reestablish physiologic, functional range of motion. Surgical efforts must be directed at correcting specific anatomic defects and must be considered in light of the consideration that arthrotomy itself may negatively alter the joint.

Open surgical intervention has taken the following forms:

Arthrotomy with
- Disc
  - — plication
  - — repair
- Menisectomy
  - — alone
  - — with alloplastic interposition
    - @ Silastic
      temporary
      permanent
    - @ Proplast (now discontinued)
  - — with autogenous interposition
    temporal fascia flaps
    dermal grafts
    auricular cartilage grafts
  - — condylar shave
- Condylectomy

Total joint replacement

Arthrotomy is performed through a preauricular incision (Fig. 21.5). Access is gained inferiorly by dissecting on the deep plane of the temporal fascia. A flap is raised in the joint capsule, permitting access to the joint. The joint is distracted with a self-retaining retractor, and the joint is meticulously examined. In the absence of degenerative osseous changes, meniscal plication arthroplasty may be considered. A wedge is removed from the posterior aspect of the meniscus and the thickened portion of the retrodiscal pad. The meniscus is then sutured to the remaining portion of the retrodiscal pad. This is meant to prevent further meniscus subluxation. It is also thought that pain may be relieved by dividing the innervated posterior attachment. A high posteriorly angled condylar shave has also been advocated along with the meniscal plication procedure in an attempt to augment the joint space. This appears appropriate only in the face of degenerative bony pathology. Condylar shaves alone as gap arthroplasty have often failed to relieve symptoms. Menisectomy is considered if the disc is damaged or if it is dislocated anteriorly. Menisectomy alone is an established procedure that has been confirmed to produce good results but may fail to alleviate the symptomatology. Approximately 15 to 20 percent of patients continue to have pain, and some 35 to 40 percent are pain free, except when chewing hard foods. Also, menisectomized patients may develop chronic pain following trauma to the chin.[15] It has been noted that menisectomy may even predispose to ankylosis.[5] There is some concern relating to bony resorption of the condyle as well as the glenoid fossa, with subsequent development of posterior ramus height decrease and open bite deformity.

Condylectomy has been proposed as well, but predisposes to postoperative deviation and bite collapse.[16]

Interposition arthroplasty was designed to recreate a functional joint space of appropriate volume, and to eliminate potential problems associated with menisectomy or gap arthroplasty and degenerative changes consequent to bone to bone contact. Alloplastic materials, such as Silastic, may be affixed to the zygomatic arch, padding the glenoid fossa. Concomitant eminectomy may be performed to increase the joint volume anteriorly and minimize the possibility of impeded condylar translation. In addition, condylar shave may aid in further tailoring the joint. Alloplastic reconstruction of the TM joint has been attempted in an effort to fashion a functioning, painless joint with anatomic and physiologic characteristics resembling the normal joint. Prosthetic replacement of the condylar head has been performed, particularly in cases of TM pain associated with bony ankylosis, fibrous adhesions, or end-stage arthritis (Fig. 21.6). Interposition arthroplasty has been performed with alloplastic materials such as Silastic, dacron-reinforced silicone sheeting, ethylene, metal, and Proplast, as well as a bilaminate Teflon-Proplast glenoid fossa prosthesis.[17] A Silastic prosthesis has also been used as a condylar head prosthesis (Fig. 21.6B).[4]

Metallic Proplast-coated prosthetic implants, sometimes with glenoid fossa prosthetic reconstruction, were used to provide gliding surfaces and protection of adjacent bony structures. These have been associated with severe tissue reaction and discontinued. Histopatholog-

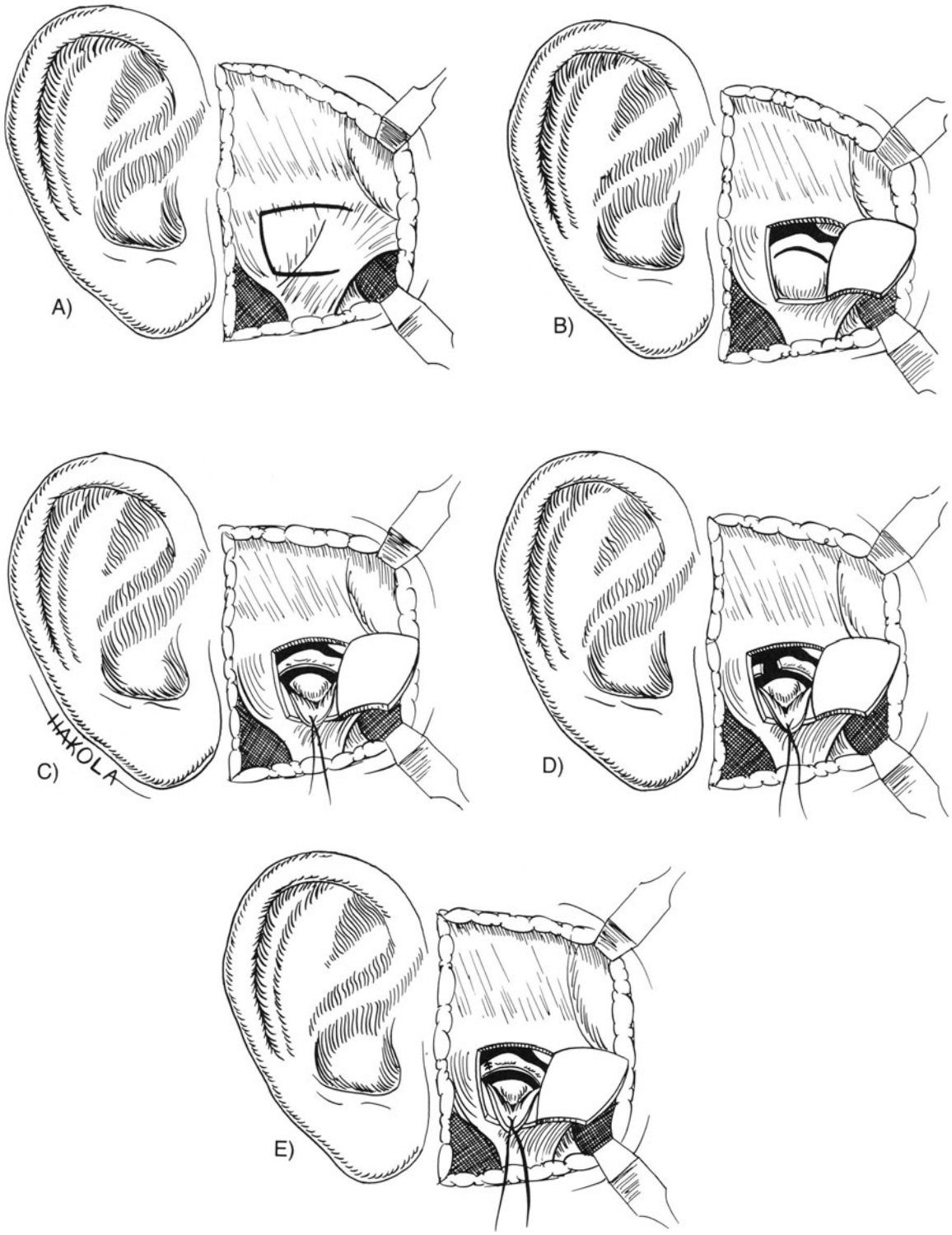

Figure 21.5. Arthrotomy is performed through a preauricular incision (A). A capsular flap is described (B). Attachments into the inferior joint space are opened (C) and the disc is isolated. A disc plication by posterior excision is shown (D,E). More commonly, extirpation is performed.

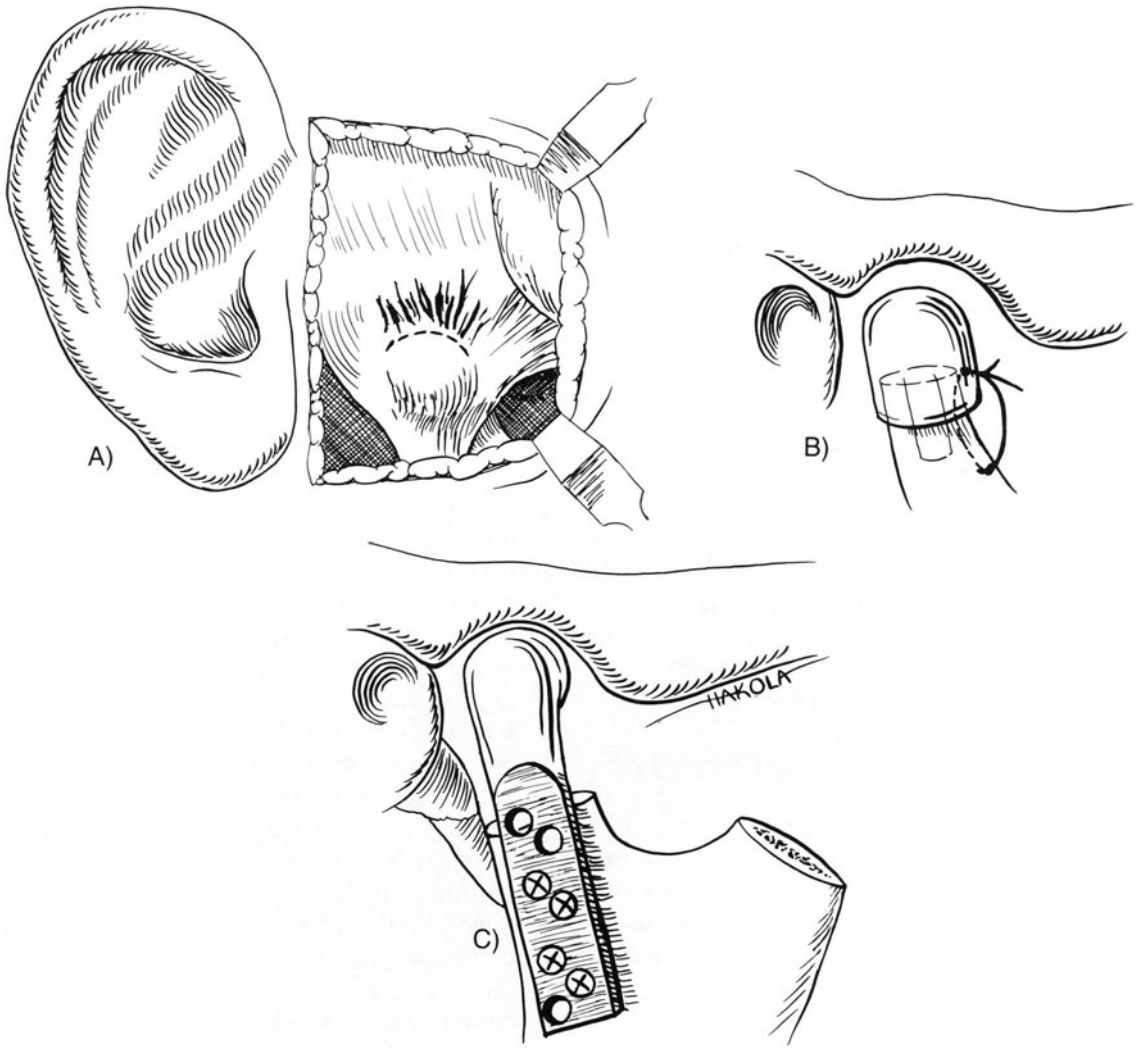

Figure 21.6. Condylar head replacement may be necessary in case of severe degenerative disease of ankylosis (A). An ulnar head prosthesis may be used when moderate joint space is encountered (B). When more significant condylar loss is seen, a variety of metal prostheses are available (C).

ically, Silastic has been associated with fragmentation and intense foreign body reaction, with multinucleated giant cells surrounding Silastic particles. Fragments of Dacron used for reinforcing the Silastic have also been found, but with less severe foreign body reaction. Regional lymphadenopathy has been seen, and lymph node biopsy has demonstrated foreign body granulomas surrounding metastasized Silastic particles. Silastic fragmentation relates to the degree of articular surface damage and irregularity. It does not endure the load of mandibular function well. Teflon-Proplast prosthetic implants were used with the intent of eliminating the risk of silicone synovitis. These implants became somewhat fashionable with reports indicating favorable results. However, these implants have resulted in significant postoperative pain (95 percent), malocclusion (30 percent), and limitation of opening (70 percent), as well as radiographic evidence of bony degenerative changes, more advanced than preoperatively (condyles: 100 percent; fossae: 60 percent).[18–20] Significant implant breakdown under the load of mastication has been seen. Giant cell reaction occurs with particles of Proplast

found in adjacent lymph tissue. There are reports of middle cranial fossa perforation by a Teflon-Proplast postmenisectomy implant.[17]

Bioceramics, such as compact aluminum oxide, have recently been employed as condylar head replacements in cases of ankylosis. This application of compact biocompatible ceramics in mandibular, TMJ, and maxillofacial reconstruction was derived from the use of porous ceramics in preprosthetic surgery. Compact aluminum oxide ceramics were reported to have compared favorably to chromium-cobalt-molybdenum alloys in resistance to corrosion and pressure.[21]

As there is still no alloplastic TMJ implant that is universally safe and effective, an interest in practical, effective autogenous implant strategies developed.

Fat grafts, dermal grafts,[22] fascia lata grafts, free pericranial grafts,[16] bovine collagen,[23] sternal cartilage, lyophilized cartilage, lyophilized dura, cryopreserved cartilage and freeze dried dura,[24] auricular cartilage grafts,[15,25,26] and costochondral grafts,[2,27–29] as well as temporalis fascia and muscle interposition flaps,[3,4,8,13,14,27,30,31] have been proposed and employed for the reconstruction of the TM joint.

Autogenous tissue for interposition arthroplasty has been proved relatively successful.

The ideal autologous disc replacement tissue must have the ability to withstand the masticatory load and remain viable, be readily available, easy to harvest with minimal donor site morbidity, and should be available in quantities sufficient for bilateral disc replacement, and exhibit minimal resorption following placement.

Dermis was first used in TM reconstruction in 1913. It is still employed by some and is touted as the autologous tissue best able to endure the trauma of temporomandibular joint operation.[22] Disadvantages of this technique include the need for a distant donor site and the potential for donor site scarring. The dermis is placed as a free graft in a nonvascularized bed, and the potential for scarring and decreased mobility exists.[4] In addition, a case of painful epidermal inclusion cyst formation in the TM joint two years postoperatively has been reported.[32]

Extrapolation from the orthopedic literature has led some authors to TMJ reconstruction with allogeneic materials: cryopreserved articular cartilage for mandibular condyle articular cartilage replacement and lyophilized dura for replacement of the meniscus.[24] Reconstruction performed in this manner does not require resection of healthy mandibular condylar neck bone (as total joint prosthetic replacement does) and allows more extensive procedures to be reserved in case of graft failure. There is concern regarding the risk of developing Creutzfeldt-Jakob disease associated with freeze-dried dura placement.[22]

Autogenous auricular cartilage has been used as an autologous replacement for alloplastic implants (Fig. 21.7), such as the Proplast-Teflon condylar head prosthesis, providing pain relief and improvement in function. It is readily available, easy to harvest, is of appropriate shape, and may remain viable in the temporomandibular joint. Because of the possibility of the formation of adhesions within the TM joint, it has been suggested that the postmenisectomy placement of a temporary Silastic implant may reduce the likelihood of

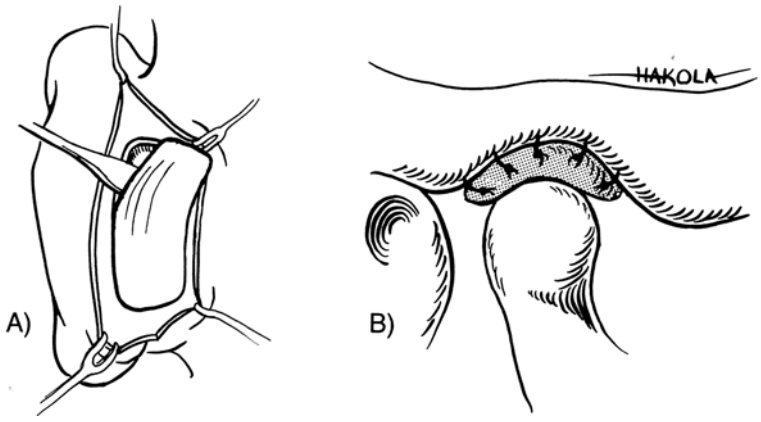

Figure 21.7. Use of auricular conchal cartilage as an interpositional arthroplasty material. (A) Graft harvested through a postauricular incision. (B) Graft secured to lateral glenoid fossa.

adhesion formation. In one study comparing interposition auricular cartilage grafting to menisectomy, however, the results slightly favored menisectomy.[15]

Pericranium, placed as a free graft following menisectomy with temporary Silastic implant placement, has been advocated. The two-stage procedure involves menisectomy and eminectomy with temporary Silastic interposition, followed 6 months later by a second stage during which the Silastic is removed and a pericranial graft is placed into the joint space. The graft is not fixed in position.[16]

A temporalis fascia and temporalis muscle flap interposition following menisectomy, usually without condylar shave, have become the most frequently performed arthroplastic procedures in the authors' hands (Fig. 21.8). Temporalis fascia has been used for repair of the retrodiscal pad, and in combination with menisectomy or high condylectomy for reconstruction in cases where the meniscus is unsalvageable (Fig. 21.8). The technique was first described in 1912 by Murphy[4] and has become popular in secondary reconstructions. The fascia is readily harvested as a pedicled flap through an extension of the preauricular incision employed in the arthrotomy.[8] The flap is well vascularized. The fascia and muscle have independent blood supplies. Donor site morbidity is low. Dissection is carried along the deep temporal fascia, thus skirting the facial nerve.[13] Composite temporalis myofascial flaps have been similarly used, especially when dead space obliteration is required, as in reconstruction of ankylotic TM joints. The temporalis fascia has also been combined with pericranium.[30] The temporalis fascia or myofascial flap may be used for support of the lateral joint capsule, as well. When the temporalis flap is used for postmenisectomy reconstruction, the medial attachments of the disc are preserved and the flap is secured to these. The capsule is finally closed to the flap. Results in disc replacement surgery have been consistently satisfactory. In one series, all disc replacement patients had an interincisal opening of 35+mm at 3 months.

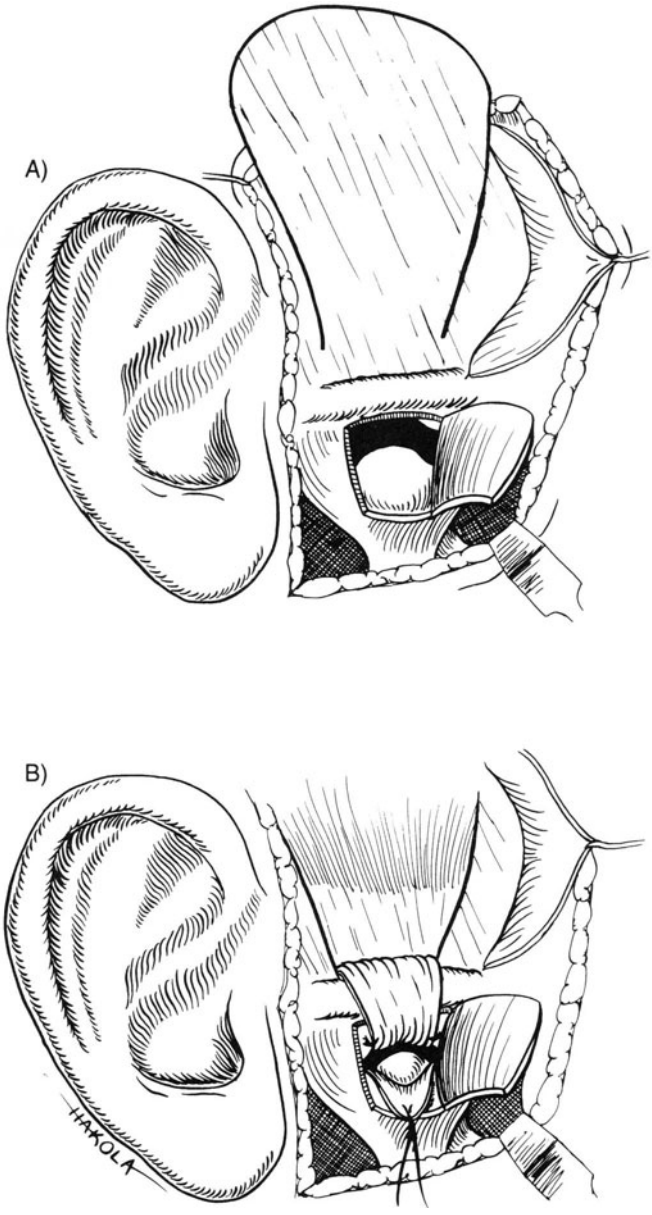

Figure 21.8. The temporal fascia flap is described for use as a disc replacement (A). It is rotated over the zygomatic arch and secured to remnants of the medial disc attachment (B).

Ankylosis

Mandibular hypomobility has been classified as true (intraarticular) or false (extraarticular).[32] Ankylosis may also be classified as bony or fibrous, according to the involved tissues. Ankylosis may be complete or incomplete. Complete ankylosis allows for maximal incisal opening of less than 5mm. The patient suffering ankylosis may experience difficulties in speech, the maintenance of oral hygiene, and chronic pain.[14] Etiologies of ankylosis include trauma, intracapsular hematomas, local or systemic infection, birth injuries, and arthritides.[14,27,33]

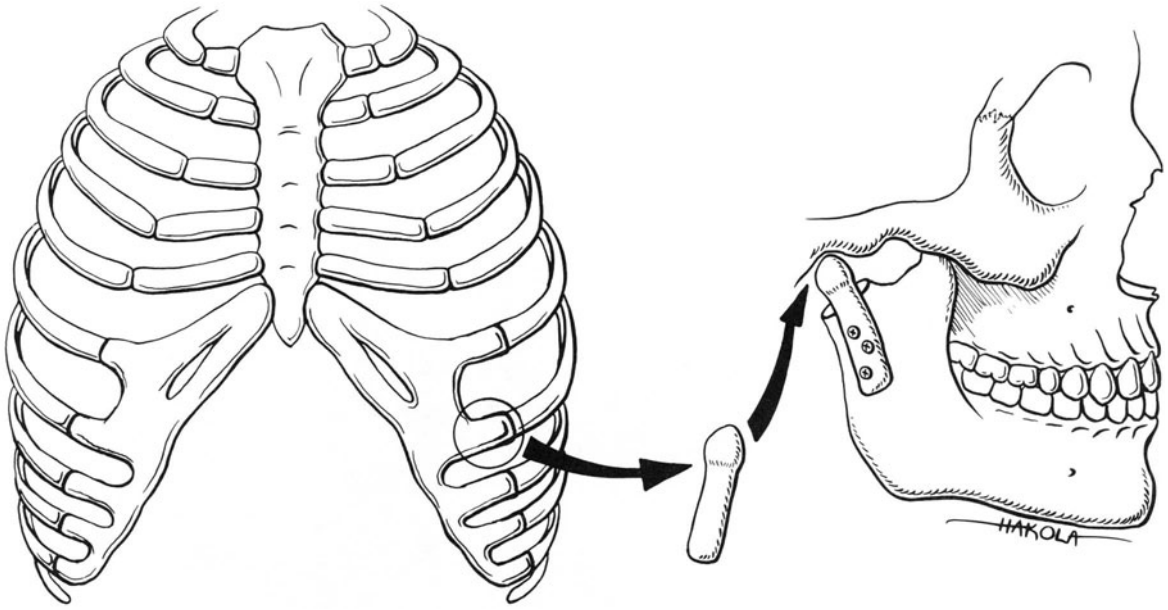

Figure 21.9. A costochondral rib graft used for condylar replacement after gap arthroplasty. (See text for details.)

In the ankylosed joint, the disc is destroyed, and the condyle and fossa cartilage are atrophied.[8] The joint space is narrowed, and thick fibrous adhesions form. As the ankylosis progresses, bony destruction, with characteristic erosions, lipping, osteophytes, subcortical cysts, destruction of the condylar contour, and finally bony ankylosis occurs. Dentofacial deformity may occur with ankylosis, especially if it is present during growth. Crossbite, incisor malalignment, Class II relationship with decreased ramus height, and anterior open bite may be seen. The patient may be able to masticate, as there is some elasticity in the mandible and the cranial sutures. Protrusive movement is impossible in true anklyosis.[8]

Multiple approaches have been used in the treatment of temporomandibular joint ankylosis. Condylectomy, gap arthroplasty, and interposition arthroplasty have been advocated (Fig. 21.6), but persistent limited range of opening has been seen. Condylectomy alone may be performed only in cases of early, fibrous ankylosis. In cases of bony fusion, there is difficulty defining the anatomic borders of the joint and glenoid fossa.[33] There is a high recurrence rate following gap arthroplasty.[5,8,33] Interposition arthroplasty with alloplastic material is widely accepted. Silastic rubber blocks[8] and acrylic resin[34] have been employed. Results, however, reveal limited range of motion despite the interposition surgery.[27] Joint replacement has been attempted to more closely approximate the normal anatomy, and maintain the increase in ramus height.[18,19,34–38] Therefore, metallic condylar prosthetic devices may be indicated in cases of ankylosis with dentofacial deformity, where concomitant joint reconstruction and orthognathic surgery are undertaken.[8] Proplast-covered vitallium implants had been used with some reported improvement in function.[19] Condylar head prostheses have been designed as intramedullary stems secured with methylmethacrylate, as in total hip replacement surgery.[37] Another prosthetic device employed in TM joint replacement is the ulnar head prosthesis.[8,14,36]

A custom chromium-cobalt-molybdenum condylar prosthesis has been used, with promising postoperative improvement in incisal opening.[38] All such devices are under scrutiny now by the FDA.

Advantages of autogenous materials in the reconstruction of the temporomandibular joint include biocompatibility, as well as the capability to undergo remodeling (Fig. 21.9). This feature is important in the reconstruction of temporomandibular joints in children.[27] According to Kaban, costochondral graft reconstruction of the TM joint must include aggressive resection of the ankylotic segment, stripping of the temporalis, medial pterygoid, and masseter muscles and scar from the ramus, and ipsilateral coronoidectomy. At this point, incisal opening is evaluated. If it is less than 35 mm, contralateral coronoidectomy and stripping of the medial pterygoid, masseter, and temporalis muscles is performed. A new joint lining is provided with a tem-

poralis fascia flap. Intermaxillary fixation with an occlusal splint is performed with a 2-mm posterior open bite to compensate for remodeling of the graft. Intermaxillary fixation is maintained for 3 to 10 days, and physiotherapy is subsequently started. There are reports of overgrowth of costochondral grafts, but this overgrowth has been lateral, not axial.[27]

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CHAPTER 22

Basic Maxillofacial Course: Laboratory Manual

Jeffrey C. Posnick and Irving M. Polayes

Outline of Laboratory Sessions

Day 1:

A. Review of Basic Dental Materials
   - Alginate, dental stone, plaster of Paris, bite wax, sticky wax, (cold cure) acrylic

B. Review of Laboratory Equipment/devices
   - Typodont
   - Dental articulator(s)
   - Face bow device
   - Arch bars

C. Laboratory Tasks

Part I

- Take alginate impressions of typodonts
- Pour up alginate impressions (of typodonts) in dental stone
- Take alginate impressions of each other (your partner)
- Take (wafer) wax bite of your partner in centric relation
- Pour-up of alginate impressions (of partner) in stone
- Mount dental casts on articulator with occlusion in (centric relation) bite
- Construct occlusal-lingual acrylic splint for mandible

Part II

- Demonstration of partial tray technique for management of multiple (segmental) mandibular jaw fracture. Explanation on how to construct a splint for a badly comminuted dental arch
- Section mandibular stone model (using coping saw or electric drill) into pieces to simulate segmental jaw fracture
- (Sticky) wax the segments against the intact upper (maxillary) arch to recreate the "preinjury" mandibular arch form. Remove several teeth from stone models to simulate edentulous spaces
- Pour up a new stone base for the mandibular segments that are being held together against the maxillary arch (teeth). The preinjury mandibular arch form is now recreated
- Construct an occlusal-lingual acrylic splint on the recreated mandible that could be reset to stabilize the fractured segments of the lower jaw.

Day 2

Demonstration of:

Part I

- Interdental and intermaxillary wiring techniques
  Application of intermaxillary wires and the use of elastic bands
  Use of acrylic for immediate intraoral splinting of jaw fractures

Part II

Construction of a Distal-Extension Splint using an acrylic base plate technique

- Remove mandibular molar teeth from typodont (cover tooth sockets with tin foil)
- Make an acrylic base plate on the mandibular part of the typodont
- Mix up acrylic dough and then place over edentulous areas of acrylic base-plate (like a bite-block)
- Lubricate upper teeth with vaseline and bring the maxillary and mandibular teeth into occlusion through the soft acrylic (this will establish an occlusion over the base plate)
- Trim excess acrylic before hardening with scalpel blade
- Once hardened, smooth splint using grinding stones or burs
- If splint demonstrates warping on the model, additional lining can be added with a soft acrylic mix
- Apply an arch bar to the constructed splint using the acrylic liquid-powder "dusting" technique

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Part III
Construction of maxillary and mandibular splints for edentulous patient (Gunning Splints)

- Remove all remaining teeth from typodonts (cover over tooth sockets with tin foil)
- Make a thin "pancake" (of acrylic dough) and drape over the maxillary stone cast to make a base-plate
- Trim excess acrylic with scalpel before it hardens
- Construct an acrylic bite-block (1/2 inch high) on the maxillary base-plate; flatten the base of the soft acrylic bite-block and allow to harden
- Construct a mandibular base-plate using the same technique
- Place Vaseline on the upper acrylic hardened bite-block and close it down onto the soft acrylic dough of the lower base plate to register a bite (clinically, this is done on the patient's mouth).
- Add additional acrylic to the splints to smooth any rough, irregular areas on the splints
- Trim hardened acrylic with rotary burr(s) or stone(s)
- Cut an opening through the acrylic in the front section to facilitate eating and breathing
- Apply arch bars to splints using the liquid-powder "dusting" technique

Part IV
Discussion of fixation techniques of constructed acrylic splints to jaws

- Circomandibular wires
- Transosseous fixation
- Suspension of skeletal fixation wires to skull
- Kirschner wires for maxillary splints

Part V
Repair of broken denture(s) to be used as Gunning splints. Piece broken dentures together as accurately as possible.

- If both upper and lower dentures are available, place into occlusion with each other
- Once fragments are accurately positioned, "sticky" wax them together
- Use each denture as if it were an alginate impression and pour dental cast in to stone
- Once stone is hardened, articulate the models and modify dentures as desired or construct new ones

Materials for Basic Maxillofacial Laboratory

- Alginate impression material
- Plaster of Paris (powder)
- Dental stone (powder)
- Dental utility wax
- Multipurpose (cold cure) acrylic
- "Sticky" wax
- Vaseline (for coating models)
- Soft bite wax
- Plasticine

Equipment and Supplies for Basic Maxillofacial Laboratory

- Plastic perforated impression trays (small, medium, large)
- Mixing bowl and spatula
- Arch bars for application of IMF
- Small electric hand drill with bits, stones, etc.
- Dental wax spatula
- Sharp utility knife
- Dental plaster knife
- Dental articulator on which to mount models
- Face bow transfer device
- Vibrator for pouring stone/plaster
- Electric grinder wheel

Basic Maxillofacial Laboratory Techniques

Background

Fractures involving the mandibular and maxillary often occur in edentulous areas where involved teeth have been previously extracted or were lost as part of the recent injury. To anatomically reduce these fractures and then maintain adequate immobilization with intermaxillary fixation (assuming plate and screw fixation is not preferred), it may be necessary to construct an acrylic splint against which the opposing teeth will occlude. Arch bars are fixed to the existing dentition or placed directly on the splint prior to inset in the mouth.

A laboratory exercise has been designed to acquaint the surgeon with the simplest methods available of acrylic splint construction. Once the basic techniques are mastered, the learned splinting techniques can be modified to manage the specific clinical problem at hand. A typodont is provided on which impressions can be taken in alginate and then stone models constructed from the alginate impressions. The teeth of the typodont can be selectively removed to simulate edentulous or partially edentulous jaws. Using this technique, various clinical situations can be simulated and splinting techniques demonstrated.

Description of Alginate Impression Taking; Pouring the Impressions in Stone; Taking a Bite Registration; Mounting Dental Casts on the Articulator; and Constructing Acrylic Splints

General Information

Dental splints can be useful to provide stability for the reduction of maxillary or mandibular fractures. Only

clinical judgment can help the surgeon decide whether splinting is a preferred option when immobilization of a particular fracture is required. There are certain clinical situations where a constructed acrylic splint may provide a useful role. A dentition with poor interdigitation may grossly appear to be satisfactory but the cusps of the teeth may be tilted too far lingually. If IMF is selected as a close reduction technique without the use of the splint, then lingual tipping with malunion might result.

Alginate Impression Taking

A familiarity with the materials, supplies and equipment needed to take dental impressions is important not only for maxillofacial fracture management but also for orthognathic surgical diagnosis and planning.

When taking alginate impressions, the impression tray size and shape is important. The tray can be rimmed with utility wax, which will help confine the impression material and give more comfort to the patient. Impression material is then measured out (directions are on the container), and put into the measured-out water within the rubber mixing bowl. By placing the powder into the liquid, there is less chance of air bubbles forming. Liquid and powder should be spatulated as if one were smoothing out soft butter (without whipping action) as this will also limit air bubble formation.

The patient who is to have alginate impressions taken should be seated in a relaxed forward position and instructed to breath slowly through his or her mouth after the tray is inserted. The setting time for the impression material is approximately 3 minutes, or when it becomes firm and rubbery. To remove the tray, after the impression material has set, simply break the suction seal with a finger placed in the retromolar area. Once removed, dry the alginate impression using compressed air or by blowing a hard stream of air on it.

Pouring Dental Impressions in Stone

When pouring dental impressions in stone, the material placed into the alginate impression is either dental plasters dental stone or some combination of the two. Once hardened, the dental stone is harder than dental plaster, and there is less chance of breaking the teeth when later removing the hardened cast from the alginate impression.

Once the plaster (or stone) has been mixed with water in a mixing bowl, you are ready to "pour up" the alginate impression in stone. To avoid or minimize trapping air between the stone and alginate interface, start by pouring small amounts of the mixed stone (or plaster) into the impression while vibrating the impression tray on the vibrating table. This allows the air bubbles to come to the surface. The object is to fill the alginate impression from the occlusal surfaces upward to avoid trapping air bubbles that could distort the cast. Professional dental laboratories or small laboratories within most dental offices have electric vibrators for assistance.

Allow the dental models to "set" or harden. If you need to construct the splint immediately, then allow the models to set for a few hours, or preferably overnight. The harder the models are at the time of separation of the models from the alginate impression, the less likely you are to fracture them. You should wait at least 30 minutes before separation.

Taking a Bite Registration

A bite registration is taken in the patients mouth and used to mount the dental models on the articulator as close to the patient's actual "centricrelation" bite as possible. One way to take a bite registration is by using a single layer of thin, pink-base plate wax and cutting the wax to fit the patient's mouth (arch form) extending just beyond the occlusal bite plane. The wax is then warmed over a Bunsen burner (or hot water) and the patient is then instructed to bite down firmly into the soft wax. It is important for the surgeon to guide the patient's condyles (to seat them) in the glenoid fossa at the time of biting (centric relation). This allows for a consistent relationship of the upper and lower teeth as they occlude. Allow the wax to cool to room temperature and carefully remove without distortion. Place this wax bite on the occlusal surfaces of the poured models and bring the models together, allowing the cusps of the teeth to occlude snugly into the impressions made in the wax bite. The dental models are now ready to mount on an articulator.

Mounting Dental Casts on an Articulator

It is important to maintain the models occluded in centric relation with the wax bite in between, while mounting them on the articulator. They are simply held together using several rubber bands. When mounting the models on a hand-held articulator (without a face bow transfer), the base of the models may be scored (or scratched) to provide better surface adhesion with the plaster used to hold them on the articulator. Saturate the models with water, mix up additional plaster, and mount each dental model on the articulator. When the mounted plaster is hard, cut the rubber bands and remove the wax bite. The mounted models are now ready to have an acrylic splint constructed on them.

Note: If using a semiadjustable articulator and if condylar relationships are important, then a face bow transfer should be carried out.

Construction of Acrylic Splint

Cold cure (room temperature setting) acrylic is most frequently used to construct a splint. Grease the models with Vaseline to prevent adherence of the acrylic to the models. Block out any undercuts with plasticine to prevent locking of the acrylic into undercuts on the model, as this would prevent removal after the acrylic is hardened.

Mix the acrylic powder and liquid as per directions on the container. When the acrylic mix becomes tacky, roll it like a piece of dough with your vaseline-greased fingers into the shape of a thin cigar. The rolled acrylic is next placed on the occlusal surface of the articulated dental models and spread over the occlusal, occlusal-lingual, and/or occlusal-buccal surfaces depending on the type of splint you are constructing. Occlude the upper and lower articulated models firmly into the soft acrylic (which has been placed over the occlusal surfaces) to reproduce the patient's bite. Trim away excess acrylic with a scalpel blade before it hardens. The more trimming that is done at this stage, the less grinding of the hardened acrylic will be necessary later. Experience will allow fairly accurate splint construction while the material is still soft.

When the splint is set (hard), remove it from the model and trim it to the desired size and shape using dental burrs and stones. Polishing the splint is effectively accomplished on a large, electrically driven pumace wheel. If this is not available, adequate polishing can be carried out with the dental stones and burrs of varied sizes and shapes. Do not use burrs on the occlusal surfaces of the splint as this will change the splint's fit to the patient's teeth.

If you are constructing a "gunning" type of splint, segments of arch bars can be "welded" to the splint by adding acrylic powder and liquid using a "dusting" technique to gain adherence of the arch bar segment to the underlying acrylic splint. If the splint is being constructed for an edentulous patient, a base-plate of acrylic is initially made. This is done by greasing (with vaseline), the edentulous model and placing a "pancake" of acrylic "dough" over the edentulous region. The acrylic base-plate is formed over the edentulous dental model with finger pressure, the excess acrylic is cut away with a scalpel. The acrylic is allowed to harden and referred to as the "base-plate." Additional acrylic is mixed, and a cigar-shaped piece is formed. This is spread out over the base-plate in the shape of a bite-block. The bite-block substitutes for the missing teeth. Add the colorless liquid (from the acrylic set) to the hardened base plate before applying the acrylic "dough," to obtain a good "weld." This bite-block shaped acrylic is made approximately 1.5 cm in height and, in effect, takes the shape of a full denture without teeth. Once hardened, it is shaped and polished as previously described.

Using similar techniques, a splint is constructed on the mandibular edentulous model. Grease the already constructed acrylic "bite-block" portion of the maxillary splint with vaseline.

Make another cigar-shaped batch of acrylic dough, and place it on the lower base plate after adding acrylic liquid to the surface of the base plate. Close the maxillary edentulous bite-block splint (sitting on the articulated dental models) on to the mandibular one.

After molding the acrylic to the desired shape but while it is still soft, trim off excess dough. Allow it to harden and then finally shape it will rotary burr and stone, and then polish.

Arch bars may be welded to the "full denture" (gunning) splints. It is a good idea to cut an opening in the front to facilitate air intake in feeding. Partial edentulous splints can be made using a similar technique.

When confronted with multiple fractures in either the maxilla or mandible, segmentalization of the arches can result. A partial impression tray can be effectively used to take segmental impressions of each of the fractured and dislocated pieces. These alginate (segmental) impressions are then poured up in dental stone as independent segments. After each stone cast has hardened, they are trimmed and placed into occlusion with the dental cast of the intact opposing jaw. (Hopefully, there will be one jaw intact). The fragments are applied one by one to the intact opposing dental arch using "sticky" wax to help hold the pieces together. After the fragments have been "sticky" waxed into occlusion against the opposing intact dental cast, a new base of dental stone is poured to hold the pieces together. This recreates the patient's preinjured arch form from the badly fractured dental arch segments.

If both arches are comminuted with displacement of multiple fragments, then this technique has very limited application.

Dental Materials

Alginate Impressions: Material

Essential ingredients: a soluble salt of alginic acid (e.g., calcium sulfate), a retarder (e.g., trisodium phosphate), and an inert filler. The soluble salt of alginic acid, when mixed with water, reacts with the reactor to form an insoluble salt, which is a gel. To prevent the premature formation of the gel before the tray reaches the patient's mouth, a retarder is added. The retarder reacts with the reactor to produce an insoluble compound. When the retarder is used up, the desired reaction begins. If the spatulation time is either insufficient or too prolonged, the strength of the impression at the time of withdrawal will be decreased.

The higher the temperature of the water, within reasonable limits, the shorter the gelation time or the alginate impression material, and conversely.

Dental Plaster: Material

Gypsum must be calcined to produce plaster of paris and kindred products. The chemical reaction is as follows: $_2CaSO_4 \rightarrow (CaSO_4)_2H_2O + {}_3H_2O$.

To obtain plaster of paris, the gypsum is calcined in open kettles at a temperature of 113 to 120 degrees C (235 to 250 degrees F), whereas hydrocal is obtained by calcining the gypsum in a closed kettle under a steam pressure of 17 pounds per square inch gage pressure, and a temperature of 123 degrees C (253 degrees F). Plaster of paris is the hemihydrate of gypsum [$(CaSO_4)_2H_2O$].

The plaster first goes into solution to saturation. The following chemical reaction then takes place: $(CaSO_4)_2H_2O + {}_3H_2O \rightarrow {}_2CaSO_4 \cdot {}_2H_2O$ + 3900 calories per gram. The gypsum formed, being less soluble than the plaster, is supersaturated in solution, and it therefore precipitates. According to one theory, it immediately forms needle-like crystals; but according to the colloidal theory, it first precipitates as a gel, in which the crystals grow from nuclei of crystallization. The reaction is exothermic.

Five factors that influence the setting time of plaster of paris:

1. Manufacturing process:
   a. Temperature of calcining—in general, the higher the calcining temperature, the slower the set
   b. The amount of gypsum left in the plaster—the more gypsum left, the faster the set
   c. Addition of adulterants
2. Spatulation time: within limits, the longer the spatulation time, the quicker the set.
3. Water-plaster ratio: the more water added to the mix, the slower the setting time.
4. Temperature of the water: the higher the temperature of the water, provided the temperature does not exceed 86 degrees F (30 degrees C), the faster the set. When the temperature exceeds approximately 120 degrees (50 degrees C), the setting time is decreased. The effect is not pronounced, however, at any normal room temperature.
5. Addition of adulterants: accelerators decrease the setting time, and retarders increase it.

Dental Stone (Hydrocal): Material

Hydrocal

This is a specially prepared plaster, the particles of which are smooth and nonporous, in contrast, to particles of plaster of paris, which are rough and porous. The result is that hydrocal can be mixed with much less gaging water than is necessary for the mixing of plaster; consequently the hardened cast will be much stronger.

Step-by-Step Technique of Basic Laboratory Skills

Taking Alginate Impressions

1. Select an impression tray that will fit the maxillary and mandibular arch form of the dentiform. Remember that the impression tray must include all surfaces of the teeth, as well as the alveolar gingiva including the labial and buccal sulcus.
2. Moisten Ivorine teeth and the surfaces of the typodont to be included in the impression.
3. The alginate scoop and water vials are marked so that one *level scoop* full of alginate powder requires one half of a vial of water (i.e., filled to 1/2 mark on vial). For an average intraoral impression of a patient, two scoops are necessary—but for the laboratory dentiform use one scoop.
4. Pour room temperature (72 degrees F) water into mixing bowl and add powder.

Note: Warmer water hastens set while colder water retards set.

5. Spatulate the mix thoroughly until a creamy consistency (40 seconds).
6. Fill tray uniformly so that no "bubbles" or air gets trapped in the mix and place over "impression zone" of teeth and gingiva, uniformly seating the tray. The impression tray must be seated in one continuous motion without withdrawing the tray.
7. Hold tray in place until mix is set (approximately 5 minutes).
8. When alginate is set, remove impression. Alginate is "elastic" and will snap out from undercut areas as it is lifted off of teeth.
9. If possible, pour stone model immediately. If pouring model must be delayed, *do not* leave impression in water but wrap in moist towel and seal in plastic bag to retain moisture.

Pouring Stone or Plaster Models

1. Place alginate impression in a moist paper towel until ready to pour up stone/plaster models.
2. Using plaster (or stone) powder and add sufficient powder to water, spatulating while adding water until a *light* creamy consistency is achieved. Vibrate the mixing bowl to ensure loss of air bubbles.
3. Hold alginate impression on vibrating table and pour in the stone mix starting from the posterior tooth imprint. Maintain a continuous stream so that it rolls around the entire impression by the *force of its own vis-*

cosity to prevent trapping of air bubbles and thus obtain a solid, uniform model.

4. Once completed allow it to set at least 8 minutes or until *exothermic* heat is noted to be decreasing.

5. Gently and carefully lift alginate impression from stone.

Note: These models will now serve as a "patient" for practice of various wiring and fracture reduction techniques.

Making Gunning Acrylic Splint

1. Take impressions with alginate material.
   a. Place the utility wax on edges of trays so as to help confine the soft impression material.
2. Pour mixed stone into the impression.
   a. Avoid trapping air bubbles by:
      1. Making smooth mix.
      2. Vibrate tray on table while pouring stone.
   b. Trim excess stone before it hardens.
      1. Model is hard enough to use in 15 to 30 minutes.
3. Grease models and hands with Vaseline. Mix up cold cure acrylic according to directions in the set. If undercuts are present on model, either fill them with wax or plasticine.
4. Make a "pancake" of the soft acrylic and drape over the model. Force it down into recesses of the model. Trim excess with knife. Acrylic will harden in about 10 to 15 minutes.
5. Remove hardened base-plate. Trim and polish.
6. Mix up some additional acrylic powder and liquid. Coat the ridge area (contact area) of the maxillary baseplate with the liquid. When the acrylic has reached the consistency of soft putty, roll it up like a thin cigar and build up a bite rim about 1 inch high. Allow ridge to set hard. Trim and polish.
7. Mix up additional acrylic for the mandibular baseplate and construct. Place acrylic on the lower baseplate in the same manner as with the maxillary (upper) splint. Now place both the upper and lower splints in the patient's mouth and have the patient close down to the desired bite with additional acrylic in between and allow to harden.

Note: Vaseline must be applied either to the upper or lower splint so that this additional acrylic adheres to only one of the splints.

8. Arch bars may be attached to splints with acrylic, if desired.

CHAPTER 23

The Application of Dental Splints in Regard to the Modern Technique of Rigid Fixation: A Basic Maxillofacial Technique Reviewed

Jonathan S. Jacobs, Stephen A. Chidyllo, and James W. Ferraro

The use of dental splints in the treatment of maxillofacial fractures has been part of the plastic surgeons armamentarium since first reported by Bunon (1743).[1] He used a block of ivory, which he wired by means of two holes into the jaw. During the 1800s Drs. Thomas D. Gunning and James B. Bean devised several types of dental splints, which were secured to the maxillofacial skeleton with an external device.[1-3] With the development of rigid fixation and the application of craniofacial techniques to the treatment of facial fractures, there may be fewer indications for their use. However, certain applications in which they can effectively be used in the definitive management of complex maxillofacial fractures are still evident. Such indications are to assist in the anatomic reduction of boney segments, they immobilize and maintain the reduction during the application of maxillomandibular and/or rigid fixation, and as a means of stabilization during the period of boney healing and rehabilitation. They maintain special importance when multiple segment fractures are sustained, especially with dento-alveolar components.[4-9]

Dental splints have been fabricated in many forms and from different materials. They can be as simple as the patient's existing acrylic dentures or as complex as custom fabricated casted crowns. The most commonly applied are fabricated using quick cure acrylic resin (methyl methacrylate) as either lingual, palatal, or intermaxillary splints.[4-9] They are designed to reduce and stabilize the fractured tooth-bearing segments to their pretraumatic relationship and to restore the skeletal anatomy, occlusion, function, and aesthetics.

Indications

Several authors have presented the indications for the use of dental splints in the treatment of maxillofacial fractures.[4-14] Dental splints should be used in conjunction with the accepted techniques of rigid fixation to assist in establishing and securing the patient's pretraumatic skeletal anatomy and occlusion prior to and during the application of plate and screw fixation. In cases in which the patient is edentulous or missing a significant number of teeth, a splint should be applied to provide interim stabilization of the fractured segments. This is done to accurately reestablish their occlusion and vertical dimension. Similarly, when there are multiple fracture lines or fractures that extend to both sides of the arch, the tendency is toward instability because of differential forces being applied on the boney segments by the muscles of mastication. In patients with severely displaced and comminuted fractures of the mandible, it is difficult to obtain and maintain a boney reduction due to the muscular forces on these segments (Fig. 23.1). A dental splint will assist in reducing the fracture(s) and maintain alignment opposing these forces, preventing rotation or displacement of the segments. In this manner

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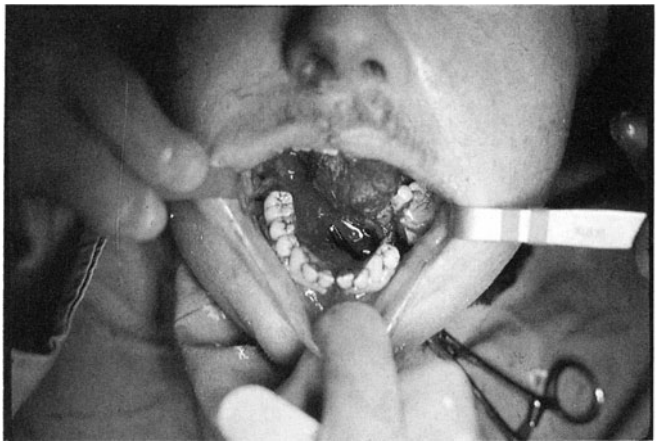


FIGURE 23.1. Twenty-two year old patient with multiple fractures of the mandible.

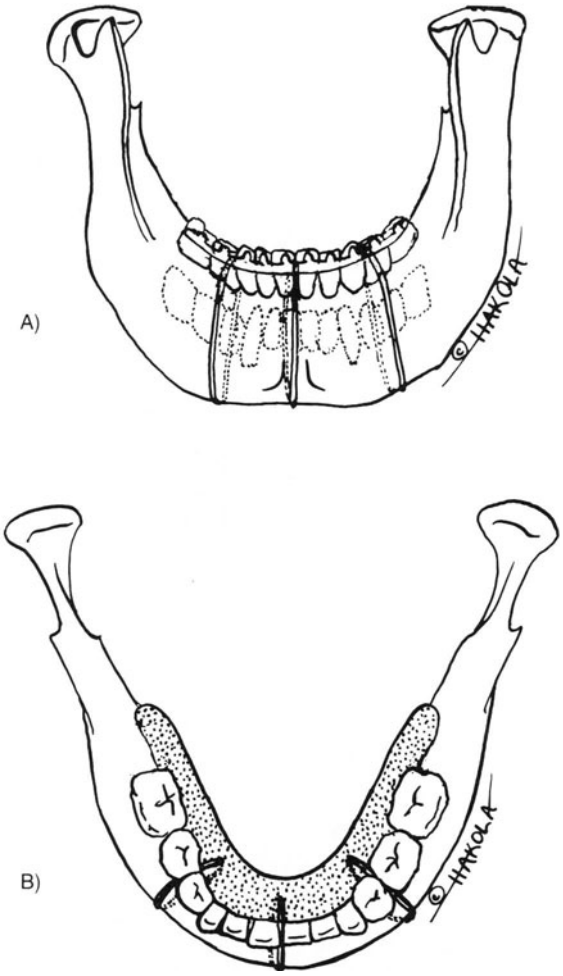


FIGURE 23.2. (A) In the mixed dentition, a splint can be secured to the mandible with circummandibular fixation without the risk of injuring the roots of development of tooth follicles. (B) A lingual splint can realign the fractured segments and maintain the reduction without interfering with the occlusion.

rigid fixation may be passively adapted to the boney segments, while the reduction is being secured by the splint.

There are several types of fractures in which conventional mandibular plate and screw fixation cannot be applied because of the risk of injury to tooth roots or developing follicles. Since it is often difficult for mandibular rigid fixation to be adapted to a fractured alveolar segment, dental splints may provide a successful means of stabilizing both the fracture and the involved teeth.[13] Mandibular fractures in children can be effectively managed with the assistance of dental splints. During the period of deciduous or mixed dentition, fractures may occur along the lines of unerupted teeth. This may result in a fracture line that is irregular, long, and oblique. Again, the use of a mandiblar plate and screws may cause injury to the developing unerupted teeth. The use of dental splints in these patients can reduce and stabilize the fracture segments without concern for damaging the developing tooth follicles (Fig. 23.2).[4–6,9,12,14]

Dental Impressions

The initial requirement in the fabrication of a dental splint is obtaining an accurate impression of the patient's mandibular and maxillary arches. Care must be taken in securing the impressions accurately, because they provide the foundation for the construction of the dental splints. An impression of the opposing arch is always required to assist in reassembling the fracture segments of the models in occlusion.

Alginate is a commonly utilized impression material. A sectional impression technique may be required when there is excessive mobility of the fracture segments. Sectional impressions are taken individually on either side of the fracture line, and the arch is reconstructed off the individual dental models. The various fractured dentoalveolar segments are assembled to the opposing dental arch using dental wax, plaster, and stone.

Dental Cast Models

Dental stone or plaster is poured into the impressions to create models of the dental arches (Fig. 23.3). When the fractures have caused derangement of the arch form, cuts are made in the model which correspond to the fracture lines (Fig. 23.4). The sections of the cast are assembled to the opposing nonfractured arch (Fig. 23.5A). The fragments are reconstructed and attached together with wax, and a new plaster base is applied to the cast (Figs. 23.5B and 23.5C). A duplication of this reconstructed cast is created on which the dental splint is fabricated

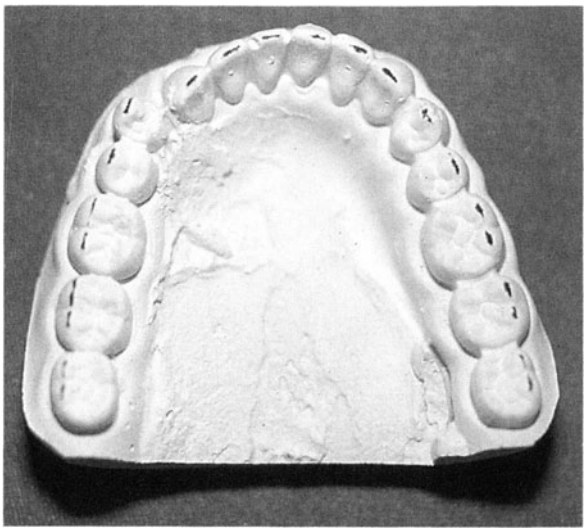


Figure 23.3. Impression of the mandibular arch with a fracture malalignment between the right cuspid and the right first bicuspid. Note the loss of the smooth arch form which is demonstrated with marks on the occlusal and incisal edges.

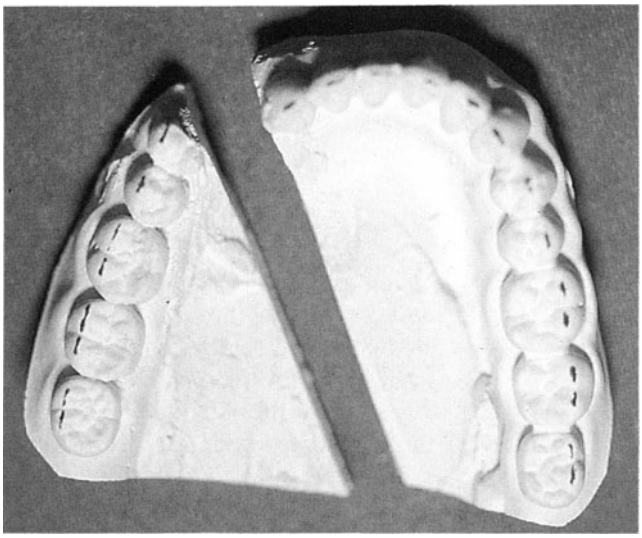
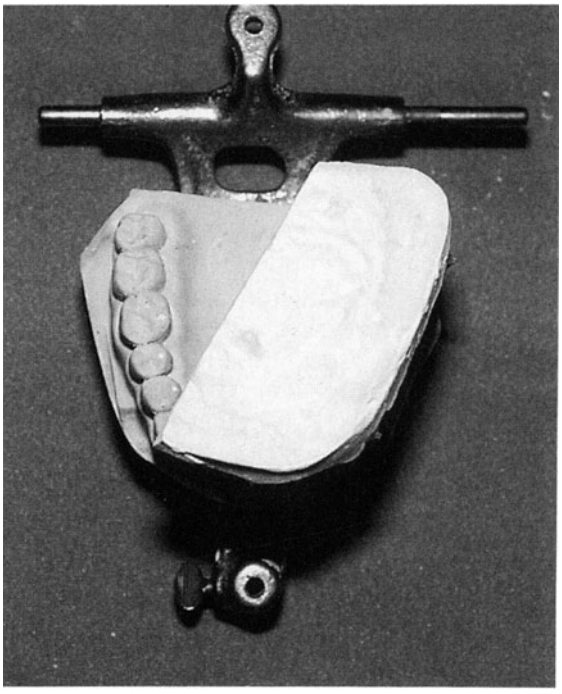


Figure 23.4. Model is sectioned at the area of fracture.



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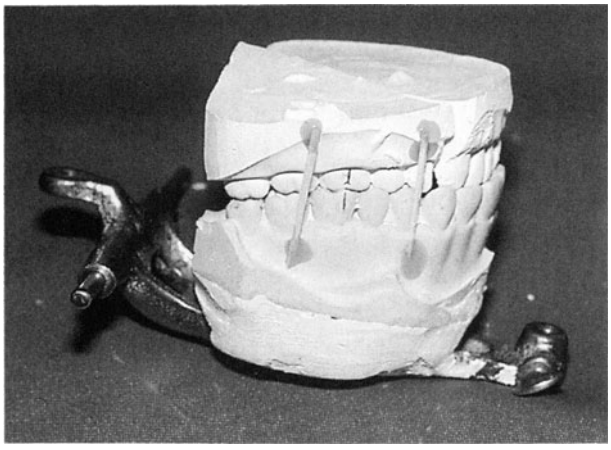
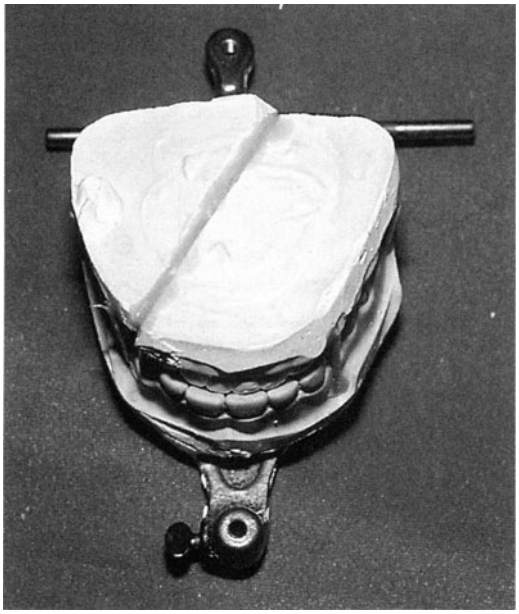


B)



C)

Figure 23.5A–C. Sectional models are fit into the maxillary occlusion and luted to the maxillary model with sticky wax to realign the dentition and reestablish arch form.

after articulation of the model to the newly fractured arch. A knowledge of dental anatomy and occlusion is necessary. The position of the occlusion at the canine and messiobuccal cusp of the first molar teeth is most pertinent.

Applied Dental Anatomy and Occlusion

Angle defined occlusion as "the normal relations of the occlusal inclined planes of the teeth when the jaws are closed."[15] He classified occlusion on the basis of what he considered to be optimal morphological relations between the maxillary and mandibular teeth. Occlusion can be simply thought of as the contact between the maxillary and mandibular teeth in all mandibular positions and movements. Occlusion has a physiological and functional component, interrelating the dentition, muscles of mastication, temporomandibular joint, and complete masticatory system into a balanced pain free state. A physiological occlusion is one in which the components function efficiently without pain and remain in a stable state of health. Few people have an ideal occlusion as described by Angle; however, most have a physiological or functional occlusion.[16,17]

Various terms are applied to the physiologic, nonpathologic position of the condyle within the glenoid fossa when the teeth are in occlusion. These include centric occlusion (CO) and centric relation (CR). CO is usually described as the bite when the teeth are in their maximal interdigitated position. CO gives no indication of condylar position. CR, on the other hand, is a term used in conjunction with a description of the condyle position. It is the most posterior unstrained position of the condyle in the glenoid fossa, which should occur simultaneous to maximal dental interdigitation. CR is a mandibular condylar position that is consistently reproducible on the patient.[18] CO and CR are not necessarily the same mandibular position, but we must attempt to have the condyle in the unstrained position at the same time as the occlusion of the dentition is in maximum bilateral contact.

The constancy of the dental cusps to fossae relationships is important to understand to determine the patient's centric occlusion (maximal intercuspal position). In closure, the buccal cusps of the mandibular posterior teeth distal to the canine are normally seated in the central fossae of the maxillary teeth, and the lingual cusps of the posterior maxillary teeth are seated in the central fossae of the mandibular teeth. These cusps are called the supporting cusps (maxillary lingual and mandibular buccal). The actual contact points of the cusp–fossae are called the centric stops or holding contacts, because they hold the teeth in a stable position (Fig. 23.6).[16] Their contact relations change with wear of the dentition. With advancing attrition, the supporting cusps seat closer to

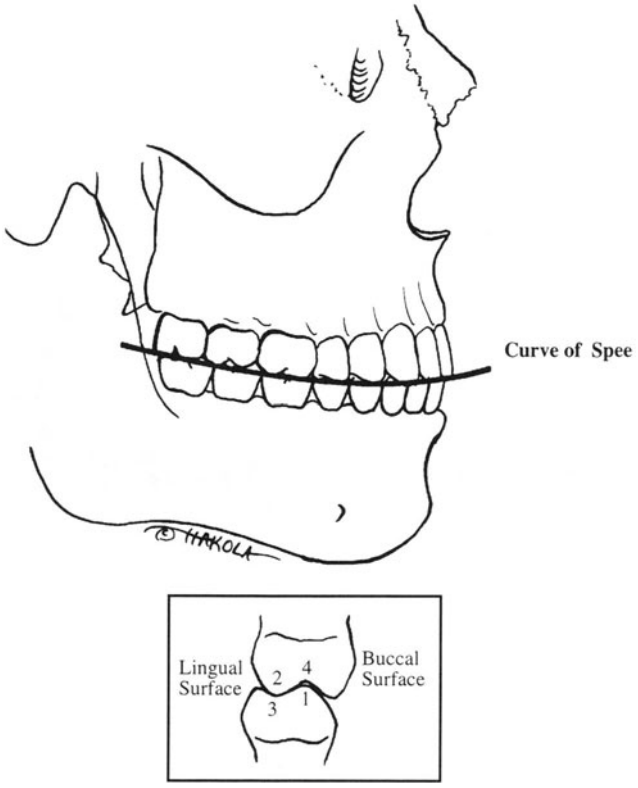


FIGURE 23.6. The mandibular buccal and maxillary lingual cusps (1) and (2) are supporting cusps. The opposing contact points (3) and (4) are central stops or holding contacts. The curve of Spee (C.S.) provides balance occlusion during mandibular function.

the depth of the opposing fossae. In reassembling models, their pretraumatic anatomical relationship may be determined by a careful evaluation of the occlusal surfaces of the teeth, in particular the facets worn on the teeth by attrition. One should match the wear facets of the cusps with those of the corresponding opposing fossae. Angle's classification of the patient's occlusion should be clinically determined and used as a guide in mounting of the casts.

Design of Dental Splints

Lingual Splints

Lingual splints are recommended for use in the mandible for treatment of complex, unstable, bilateral fractures or fractures of the symphysis or alveolar ridge. The lingual splint can help prevent inward tilting of the bony segments and counteract the tendency of the inferior border to become distracted (Fig. 23.7). Stabilization of the splint is achieved by either circum-mandibular or circumdental fixation. It is important that the splint is ligated below the height of contour of the teeth. If not, circumdental wires may become unstable and the splint displace occlusally. If there are long edentulous regions, a block of acrylic may be placed between the teeth for

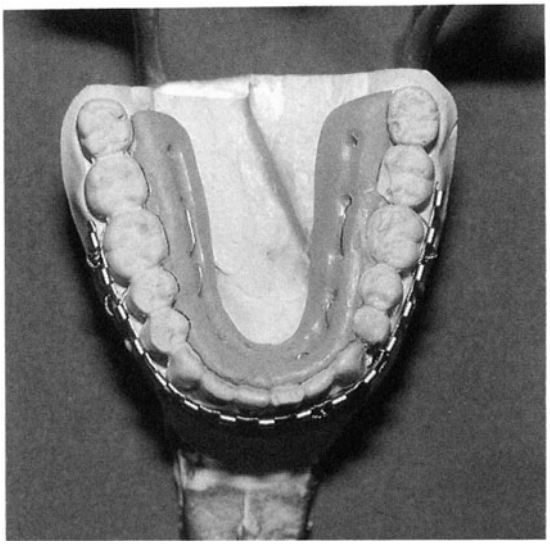
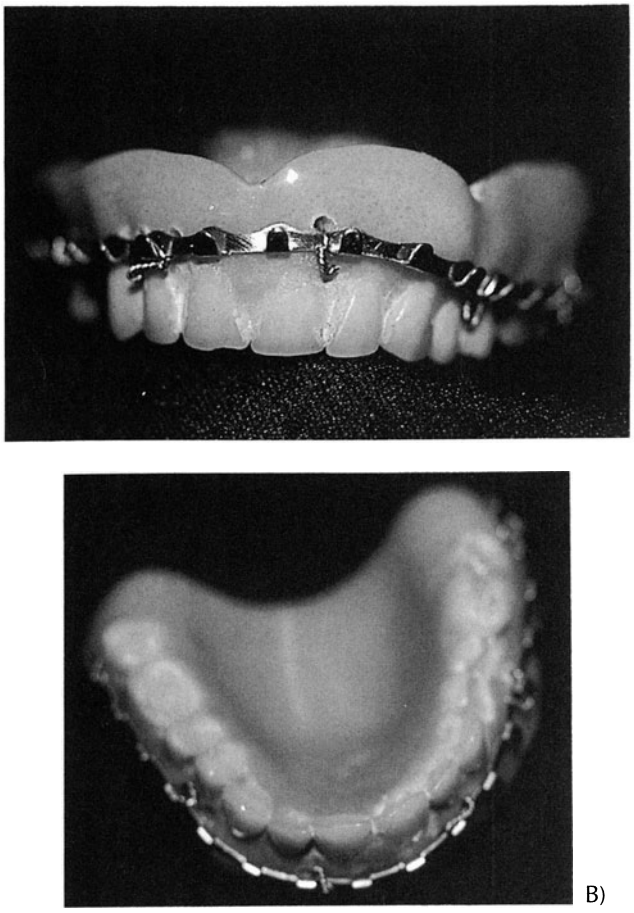


FIGURE 23.7. Finished realigned model positioned on the articulator. A lingual acrylic splint has been fabricated to help hold the aligned fracture segments.

stability. However, it should not be placed over the occlusal surfaces of the teeth. If the splint interferes with occlusion, it may open the vertical dimension and may affect the reduction. A natural curve exists in the shape of the dental arch (curve of Spee), which should be reproduced with the aid of the splint (Fig. 23.6). If added strength is desired, a metal bar may be inserted into the lingual splint during fabrication. The splint should be used in conjunction with a bucco-labial arch bar. This will provide additional support to the fixation.[4–12] If the application of rigid fixation is indicated, it can be easily applied after all of the bony fragments are anatomically reduced with the aid of the splint. The final fixation of the splint should not be completed until after the anatomic reduction of the bony segments has been performed. The lingual splint allows some leeway in order to accomplish this.

Intermaxillary Splints

Intermaxillary splints are stabilized to both the maxilla and the mandible, and are subsequently secured to one another. If the patient wears dentures—either partial or complete—they may be utilized as intermaxillary splints in the treatment of the patient's fractures (Fig. 23.8). Dentures may assist in reestablishing the pretraumatic centric occlusion of the dental arches. Arch bars are secured to the dentures prior to stabilization. Skeletal fixation of the mandibular denture is obtained by circum-mandibular or circumdental wires. The maxillary denture is secured by circumdental, piriform aperture, orbital rim, circumzygomatic, or anterior nasal spine suspension wires. A favored method is by transalveolar



A)

B)

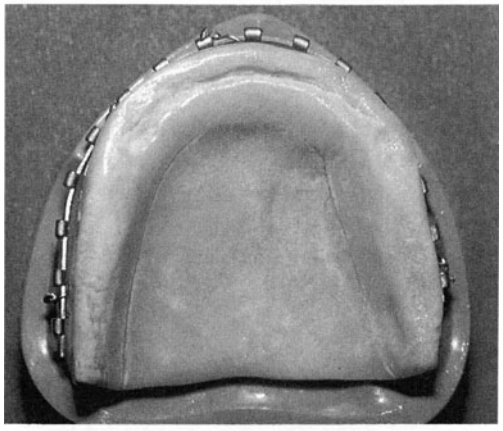
FIGURE 23.8A–B. Upper denture used as a splint with the arch bar attached to the acrylic with wires.

Steinman pins or Kirschner wire or lag screw fixation. After the skeletal fixation is established, the arches are brought into occlusion and the dentures are secured together with wire ligatures about the attached arch bars.

In the case of an edentulous patient for whom there are no dentures available to utilize as splints, partial lower full upper splints and modified Gunning splints can be fabricated to assist in the reduction and stabilization of the fractures (Figs. 23.9 and 23.10). These splints are fabricated off an acrylic base plate that fits intimately over the edentulous arch. Acrylic rims are placed as occlusal stops to maintain the patient's vertical dimension. An anterior opening can be placed in the splint, allowing the patient to ingest a blenderized or liquid diet. The splints are secured by skeletal fixation and subsequently wired together.

These splints may be used in conjunction with rigid fixation or as an interim stabilization prior to the placement of plate and screw fixation. The splints may be immediately removed if the fractured segments are stable, or they may used to provide additional temporary support. In general, placement of splints over fresh incisions or lacerations should be avoided.

A)



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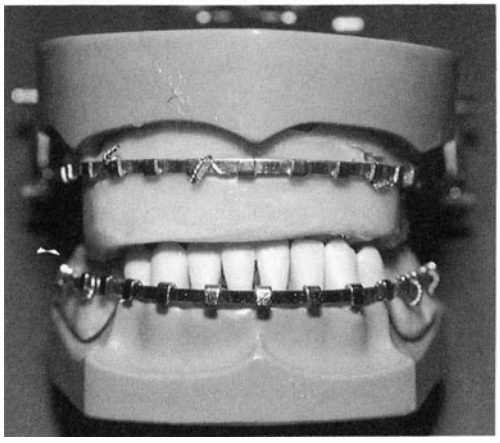


C)

FIGURE 23.9A–C. Partial lower splint and full upper splint with arch bars attached. These can be secured to the maxilla and mandible with suspension wires. Arch bars are then used for intermaxillary fixation.

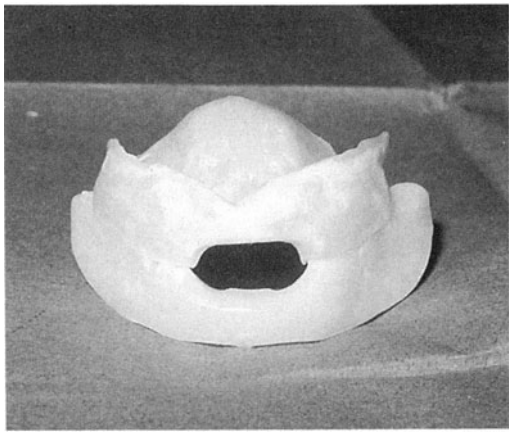
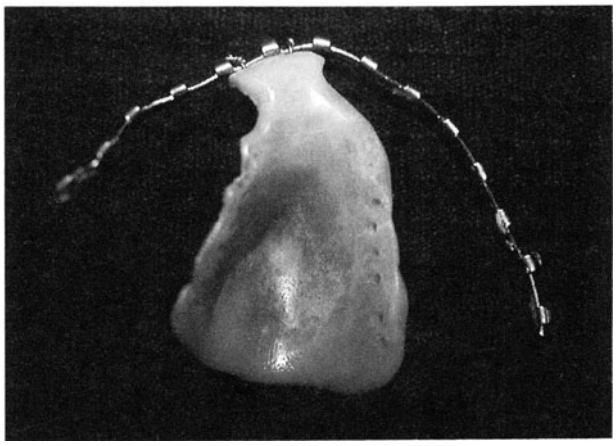
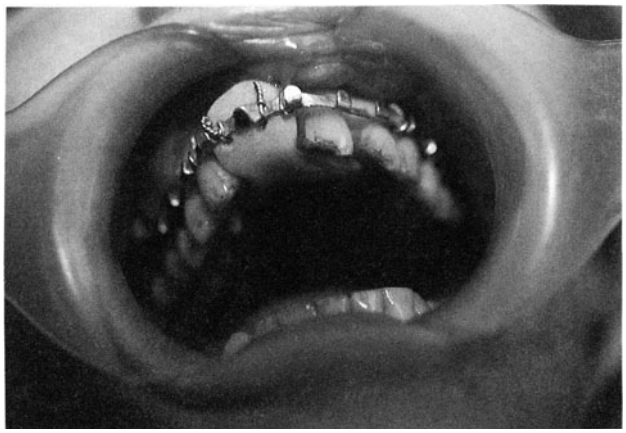


FIGURE 23.10. Gunning splints can be secured with circummandibular wires and suspension wires from the maxillary tuberosity or the zygomatic arches.



A)



B)

FIGURE 23.11A–B. A palatal splint with the arch bar attached. It is secured to the palate by attaching the arch bar to the maxillary dentition.

Palatal Splints

Sagittal fractures of the maxilla with dento-alveolar components may be reduced with the assistance of a palatal splint. The splint is inserted off the occlusal surfaces, and the individual bony fragments reduced (Fig. 23.11). Skeleton fixation is achieved as previously described. The splint stabilizes the fractured bony seg-

ments either during the application of maxillo-mandibular or rigid fixation.

It is important that these splints be constructed with palatal relief, contacting only the palatal surfaces of the maxillary teeth. This is ensured by placing a thin spacer of dental wax or foil on the cast in the areas of desired relief prior to the fabrication of the splint. If excessive gingival or palatal contact is allowed, pressure necrosis may result.

Cast Crown Splints

These splints consist of casted crowns that are attached together and intimately fit over the existing dentition. The splint is cemented in place over the crowns of the teeth. Their use has been reported in the treatment of mandibular fractures in patients with short crowns, dento-alveolar fractures, periodontitis, and in children with a deciduous or mixed dentition.[4–6,12,13]

Discussion

The treatment of maxillofacial fractures has evolved over the years from simple stabilization using external bandages and prolonged immobilization, to early mandibular function using craniofacial surgical techniques and rigid fixation. The need for dental splints in the treatment of facial fractures has decreased, and their indications limited. They are rarely indicated in the treatment of simple fractures of one arch when separate dento-alveolar components are not present. However, there are several situations in which a splint will provide assistance in the management of maxillofacial fractures. Splints are used to reduce and stabilize the fractured segments, while preventing distraction and displacement during the application of rigid fixation.

In the management of combined fractures of both the maxilla and mandible, we recommend that the fracture of the least comminuted arch be reduced first. Subsequently, the opposing arch may be reconstructed using this skeletal and occlusal relationship as a foundation. An alternative acceptable technique is with staged operative procedures in which the less comminuted fracture is reconstructed first, a new impression and dental model of this arch performed, and the opposing more comminuted arch is subsequently reconstructed to this new arch form with splints in a delayed operative procedure.

Interocclusal splints are contraindicated in the treatment of comminuted maxillofacial fractures. The use of such a splint alone may not prevent distraction, labial lingual tilting, or rotation of the fractured segments. Its use may complicate the treatment of mandibular fractures by causing distraction of the fragments at the inferior border. These splints are effective in elective cases in which a specific predicted occlusal relationships between the maxillary and mandibular arches is to be created during surgery.

There are few contraindications to the use of dental splints. They should not be used if simple wiring or maxillo-mandibular fixation will suffice. If time is a prime consideration, or if the bulk of the splint will interfere with the patient's function.[4] The type of dental splint that is utilized will be determined by the clinical state of the patient's dentition, the radiological evaluation, available laboratory facilities, and experience of the surgeon.

Conclusion

Dental splints still play a pertinent role in the treatment of maxillofacial fractures, even with advancements in the techniques of craniofacial surgery and rigid fixation. They are still important in the treatment of complex facial fractures. Splints assist with the reduction and stabilization of the fracture segments prior to the application of maxillomandibular and plate and screw fixation. Their application will add accuracy and hasten the treatment of complex facial fractures. Dental splinting and the ability to construct splints needs to remain as part of the basic core teachings of maxillofacial surgery. Once the surgeon becomes familiar with the use of such splints, the time required to construct them is minimal in regard to the benefit they provide.

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